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OF THE

FORTY-FIRST ANNUAL MEETING

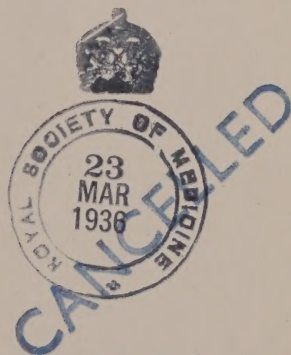
OF THE

American Laryngological,  
Rhinological and  
Otological Society,  
Inc.

HELD IN

TORONTO, CANADA

June 3, 4 and 5, 1935



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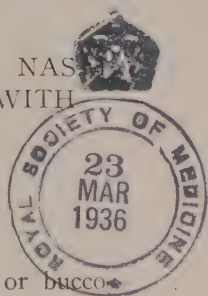
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TREATMENT OF CARCINOMA OF THE NOSE, NASAL  
SINUSES, MOUTH AND BUCCOPHARYNX, WITH  
SOME REMARKS ON DIAGNOSIS

By NORMAN PATTERSON, F.R.C.S.

London, England



When carcinoma occurs in the region of the mouth or buccopharynx diagnosis, as a rule, is a matter of no great difficulty. It is well in all cases to make investigations as to a possible syphilitic taint remembering, however, that a positive Wassermann reaction is often present in those suffering from malignant disease. Carcinomatous or more rarely papillomatous transformation may occur in a leucoplakial area. I have under my care at the present moment a man from whom I removed a large papilloma of the mucous membrane of the right cheek who presents in a corresponding area of the left cheek a patch of leucoplakia which appears in places to be undergoing a warty change. It may be very difficult to recognize that a transformation is taking place from a simple to a malignant condition, and one or more microscopic examinations may be necessary. Bowen in 1912 drew attention to what he styled precancerous dermatitis. Recently Howarth, Panton and Woods have described a similar change affecting the soft palate. Apparently the disease, although very chronic, eventually develops into squamous celled carcinoma.

Panton and Woods give the following account of the pathological appearances: "The epithelium is greatly thickened, the thickening consisting of great elongation of the interpapillary processes, the epithelium between these processes being thin. There is a layer of necrosed keratinized epithelium on the surface infiltrated with inflammatory cells and masses of microorganisms. The keratinized epithelium is continued down in the center of many of the down-



growing epithelial processes. There are small groups of large stratum granulosum cells. The cells of the stratum malpighii vary considerably in size. In the cytoplasm of many of them are eosinophile hyaline bodies; such a body frequently fills a cell, the nucleus being flattened at its periphery. The basal cells of the epidermis are in places several layers thick. They vary in size and general appearance and often have irregular shaped hyperchromatic nuclei. The cells of the basal layers of the epithelium are sometimes not sharply demarcated from the subepithelial connective tissue and fade off into the latter as ill defined irregular shaped trabeculae simulating early invading carcinoma. The subepithelial connective tissue is fibrotic and contains areas of dense infiltration with leucocytes, plasma cells and eosinophile leucocytes" (*vide Journal of Laryngology*, January, 1935).

I think we are all familiar with a tumor which affects more especially the mucous membrane of the hard palate and sometimes of the alveolus where from a much thickened epithelium project innumerable white almost filamentous processes forming an area like a thick mat. The disease spreads locally, but, at any rate for years, may not undergo malignant change. In contrast to such a condition we sometimes meet with a little roughness or local infiltration of the mucous membrane which under the microscope shows evidence of invasion of the deeper structures and presents a true picture of malignancy. If there is any doubt as to the nature of a lesion it is better to treat it as being cancerous or precancerous. If the patient is unwilling to submit to treatment he should be examined frequently. Even after the most careful inspection a carcinoma is sometimes passed over unobserved, and it is well to recognize the importance of palpation. I have on a good many occasions met with precocious metastases in the cervical glands when inspection revealed nothing abnormal in the buccopharynx. Many years ago a patient was referred to me by one of my surgical colleagues for laryngoscopy. He had a large mass of glands in the neck; no primary focus had been discovered. Nothing abnormal was visible in the mouth, pharynx or larynx, but on palpation I located a hard nodule about the size of a pea in the substance of the tongue near its edge. I have also met with small epitheliomata of the tonsil associated with massive secondary glands. In a recent article in *The Lancet* I reported two cases of this nature, one of them occurring in a woman. Some years ago a patient was brought to me with a large hard mass in the neck. As no cause for this could be discovered it was thought that the tumor might be primary in the neck. I removed the cervical tumor and under the microscope it proved to be a carcinoma. Three years later

carcinoma of the larynx was diagnosed. Recently I saw an analogous case. My friend, Colonel Tresidder, an experienced laryngologist, removed a mass from the right supraclavicular region which proved to be composed of epitheliomatous glands. The larynx appeared normal at that time. Nine months later I saw the patient in consultation and he presented a recurrence in the neck and a subglottic tumor on the right side which had all the appearances of malignancy.

Tumors at first thought to be primary and situated in regions far removed from the mouth or pharynx may prove on further investigation to be secondary. P. Turner describes a large tumor of the scapula which was regarded as a primary sarcoma; after further study of the case it was found to be secondary to a carcinoma of the tonsil. I have met with a sarcoma of the scapula secondary to a similar condition in the nasopharynx.

I think it probable that epithelioma of the tonsil not infrequently commences in the lining membrane of one of the crypts. These crypts often extend almost to the capsule. A tumor if it originates near the bottom of a deep crypt may attain considerable proportions before it causes any alteration of the mucous membrane surface. The tonsil gradually enlarges. In the early stages such a tumor would completely elude detection although carcinoma originating near the orifice of a crypt might reveal itself early to the palpating finger. In such cases if the tonsil were removed and submitted to serial section a tiny malignant focus would probably be discovered.

In one of my cases where the tonsil was sent for examination the growth was so small that the pathologist had great difficulty in its location; microscopic examination showed it to be a carcinoma responsible for a large mass of malignant glands. I think it might be a wise procedure when metastases are present in the neck, and when no primary focus can be discovered to remove the corresponding tonsil for pathological investigation. Is it possible that tonsillar sepsis or the presence of small calcarious deposits in the crypts plays any part in the development of cancer in this region?

Practically all the cases of carcinoma of the tonsil and its neighborhood which have come under my care passed through childhood and early adult life during the time when tonsillectomy was a rare operation. Would some of these patients have escaped the disease if the tonsils had been removed?

Success or failure in treatment may depend to a large extent on whether or not the disease has invaded the cervical glands; eradication of the disease in the neck is generally much more difficult to accomplish than destruction of the primary tumor. On another

occasion I have dealt more fully with the glandular problem, but I should like now to touch on some aspects of the question. The primary growth, which is single, presents as a rule a naked surface which can be examined by the observer although sometimes the tumor is almost entirely buried. Its extent, size and relationship to surrounding structures can be gauged with some accuracy. How different is the case with glandular metastases. We cannot in some cases, after the most careful search, determine the presence of infected glands even when the neck is opened at operation, and when the glands are obviously involved we have no means of ascertaining to what extent the disease is broadcast. Another difficulty is that what on clinical grounds appear to be glandular metastases may under the microscope show nothing beyond inflammatory changes. Some idea of the likelihood of metastases being present may be obtained from the site, character, relationships and histological appearances of the tumor. With regard to the site it may be stated that, unlike intrinsic cancer of the larynx, cancer of the upper jaw, interior of the nose and nasal sinuses, a tumor situated in the buccal cavity, the pharynx or any of its subdivisions is likely to be complicated by early spread to the glandular fields. The nearer the tumor approaches to the middle line the more is the likelihood of bilateral metastases.

Tumors arising in the posterior third of the tongue have a particularly evil reputation for rapid and widespread dissemination. The size of the primary tumor is of less importance than its relationship to deep structures. A tumor which presents a wartlike appearance especially if there is a tendency to sloughing of its surface may remain for some time localized as contrasted with one which penetrates into the tissues and shows no sign of ulceration. At any moment, however, a localized growth may begin to infiltrate and disseminate. Sepsis probably plays an important part and the growth may diminish in size after the extraction of diseased teeth. The glandular swellings may also subside or even disappear. There seems to be an etiological connection with sepsis which is probably one of the reasons why cancer in this region is relatively much more common among the humbler classes. The histological features of the tumor also have much to do with rapidity of growth and spread to neighboring and distant parts.

The brilliant work of Broders has thrown new light on this subject. The histology of a tumor may, however, not be uniform. In considering the extent of glandular metastases it must be borne in mind that dissemination may have reached the mediastinal or axillary



glands. Sometimes the secondary tumors tend to develop with much greater rapidity than the primary growth and in the metastatic growth there may be a relatively greater number of cells undergoing active division. In some cases it is thought that the less rapid growth of the primary tumor may be due to the fact that it is developing in an area which is precancerous and therefore prepared to offer resistance. On the other hand retrogression may take place, and Gould described a case of microscopically proved cancer of the breast with metastases in the axilla, skin of the neck, lungs and femur where complete disappearances took place, and the patient became quite well. It has been stated that carcinoma of the tongue, tonsil and pharynx show gland metastases in 80 percent of fatal cases. Distant metastases are fortunately uncommon although they are not so rare as might be supposed. Willis reports that in sixty-two autopsies on cases of epidermoid cancer of the head and neck he found invasion of the main veins in twenty-nine cases and blood-borne visceral metastases in twenty-four cases. In one case the main portal and hepatic veins were involved as well as the inferior vena cava. Invasion of arteries whether large or small is very rare. Contrasted with carcinoma of the breast, prostate, and thyroid, skeletal metastases are exceedingly uncommon. Metastatic growths have been recorded in the regions under consideration but they are of extreme rarity. Metastases in the tongue have been described secondary to primary cancer of the lung, kidney and uterus. Secondary tumors of the tonsil have been reported when the primary was in the mamma, stomach or prostate. I have seen a tumor of the tonsil which appeared to be secondary to a testicular growth. The question of removal of a fragment of the primary tumor for microscopic examination prior to operation is a somewhat vexed one; the nature of the treatment, however, may have to be modified after considering the histology of the growth. When malignant glands are removed from the neck as a first procedure a report can be obtained without interfering with the primary tumor. In removing any portion of the primary growth the diathermy knife should whenever possible be used in preference to cold steel so that any chance of traumatic dissemination is eliminated. Pathological findings are not always infallible and I have on a number of occasions, especially in nasal cases, received a report that a tumor was innocent, or of an inflammatory nature, where eventually it was proved to be malignant. Examination of the tissue removed from the neck is only of value when a positive report is obtained. A negative finding may be misleading.

To my mind the question of treatment should be approached in the reverse order to the mode of spread of the disease, and I have therefore prefaced considerations relating to the primary tumor by a few remarks on the metastatic problem which often dominates the situation. Many a failure might have been a success had the glandular extensions received the attention they deserved. It seems to be a fairly common practice to deal with the primary growth and pay very little attention to the glandular area. Since the introduction of radium this state of affairs has become worse. It has been my practice when a neck operation was part of the surgical procedure (and this means block dissection in the majority of patients), to perform this operation two or three weeks prior to the attack on the primary tumor.

Let me now consider the primary growth. For many years I have employed diathermy in its treatment. In my early cases I attempted to destroy the tumor piece meal, but soon I found this method unsatisfactory and I developed a plan of attack comparable in every respect to the employment of the knife, save that instead of cold steel, a diathermy terminal was used to remove the tumor with a sufficient margin of healthy tissue. A knife with a slightly serrated edge proved very useful for this purpose, or a needle was used. Bleeding was to a large extent avoided, the tissues became sterilized as they were divided, so preventing cell implantation. Destruction penetrated well beyond the line of section.

A few years ago like many others I practically abandoned surgery in treatment of the primary growth in cancer of this and other regions, but only for a time, in order to give my patients the benefit of treatment by radium which seemed to have such great possibilities. Often the immediate results were dramatic, but too frequently in a comparatively short time the disease returned. Hoping that improved technique would lead to more encouraging results I persevered with radium. Eventually I abandoned radium as a sole means of treatment in any but very advanced or inoperable cases and returned to diathermy as the main weapon of attack.

More recently I have been trying ray treatment in conjunction with diathermy. In any given case a decision has to be made whether to employ radiation before or after the use of diathermy, or perhaps before as well as after its use. Needles or seeds may be inserted a week or so prior to employing diathermy excision the objection of course being traumatism of the main mass of the tumor or of deep processes extending outwards beneath the surface with the consequence that malignant cells may be dislodged and travel by lymphatic

tics or blood vessels to distant parts. As an illustration of what may happen I refer to a case which came under my care when I was using radium as a routine method in the treatment of the primary tumor. A patient was sent to me from India with carcinoma of the left tonsil and a large mass of secondary glands. I carried out a block dissection and afterwards treated the tonsil area by the insertion of radium needles. Within three months the patient died of what was diagnosed by an eminent surgeon as abdominal metastases. There was no recurrence in the pharynx or neck. I think the rapid development of malignant disease in the abdomen must have been due to dissemination associated with the introduction of the needles. It seems to me that implantation of radium will before very long be discarded in favor of teleradiation or deep ray therapy.

There is something to be said, however, more especially as a bomb is not always available, for the careful insertion of radium or radon around a tumor a week or so prior to its removal by diathermy. In large apparently inoperable growths the interval may be longer; the growth meanwhile may have shrunk to such proportions that its removal is a matter of no great difficulty. An alternative method is to excise the tumor with the diathermy knife and either at the time of the operation or later implant radon seeds or needles into the walls of the cavity or around the scar when healing has taken place. Instead of employing such methods, the radium bomb or deep x-ray therapy may be used. Brilliant results have been reported by various workers from treatment solely with radiation, but there seems much to be said for a combination of surgery and radium—a remark which applies even more to the metastatic tumors than to the primary growth. In our present state of knowledge it is probably wise to follow up radical surgery of the primary and secondary tumors by radical ray treatment.

In the operation of diathermy excision, attention must be paid to the following points.

- (1) Extraction of all septic teeth if possible three weeks prior to operation on the primary tumor; this often means a very complete clearance of the mouth. As a rule the extractions should be carried out at several sittings. Two of my patients succumbed to wholesale extractions. The objection has been raised that the tooth sockets may become invaded by malignant disease. I have never seen such an occurrence, although it might be more likely if the growth was in close proximity to the alveolus.

- (2) The patient should be carefully examined by a physician whose report may modify treatment or indeed put a veto on surgery.



(3) It must be decided whether an operation is to be carried out on the neck and whether it is to precede or succeed operation on the primary tumor. As a rule I perform the neck operation two or three weeks before attacking the original growth.

(4) Is ligation of the external carotid desirable? I generally secure this vessel at the end of the gland operation. There is this disadvantage, however, that rather a long interval elapses between the ligation and separation of the sloughs following diathermy. Notwithstanding this I have rarely met with hemorrhage of any consequence when this procedure has been adopted.

(5) Is diathermy in any particular case being used with a view to a cure or only as a palliative measure? Since the introduction of improved methods of radiation most advanced cases will be treated by such methods, although it must be acknowledged that diathermy has given brilliant results in many cases which were considered inoperable.

Attention must be paid to the following points.

(1) The removal of a portion of the tongue. In this case traction must be applied to bring the growth into the field of operation. This causes elongation in the anteroposterior axis of the organ and shortening in its transverse axis. Therefore before distortion occurs the area to be removed must be marked out with the electrode. In the case of other areas very little traction is necessary.

(2) The tissues should be cut into at a distance of from one-half to three-quarters of an inch from the margin of the growth and this applies to its deeper parts and under surface as well as to the visible edge. In the case of a tumor which increases in transverse diameter as it grows downwards the mucous membrane may have to be divided at a distance of more than three-quarters of an inch from the margin.

(3) A large artery such as the lingual should be isolated and if possible ligatured.

(4) If owing to the infiltrating nature of the growth its margin is difficult to define, the normal tissues and tumor may be divided radially with the diathermy knife, so as to ascertain whether or not a sufficient margin of safety is being allowed for. If doubt still exists after excision of the block of tissue it may be cut into in several directions in order to make certain that the removal has been sufficiently radical at all points. When it is discovered that in any region the section passes too near the growth, to this region the button electrode is applied.

The line of excision passes for the most part through muscular tissue which is quite easy to distinguish from the tumor, the latter

being hard and friable. After removal of the growth I always apply the diathermy button to the whole of the wall of the cavity or the cut surface. In some cases it is impossible to accomplish a true

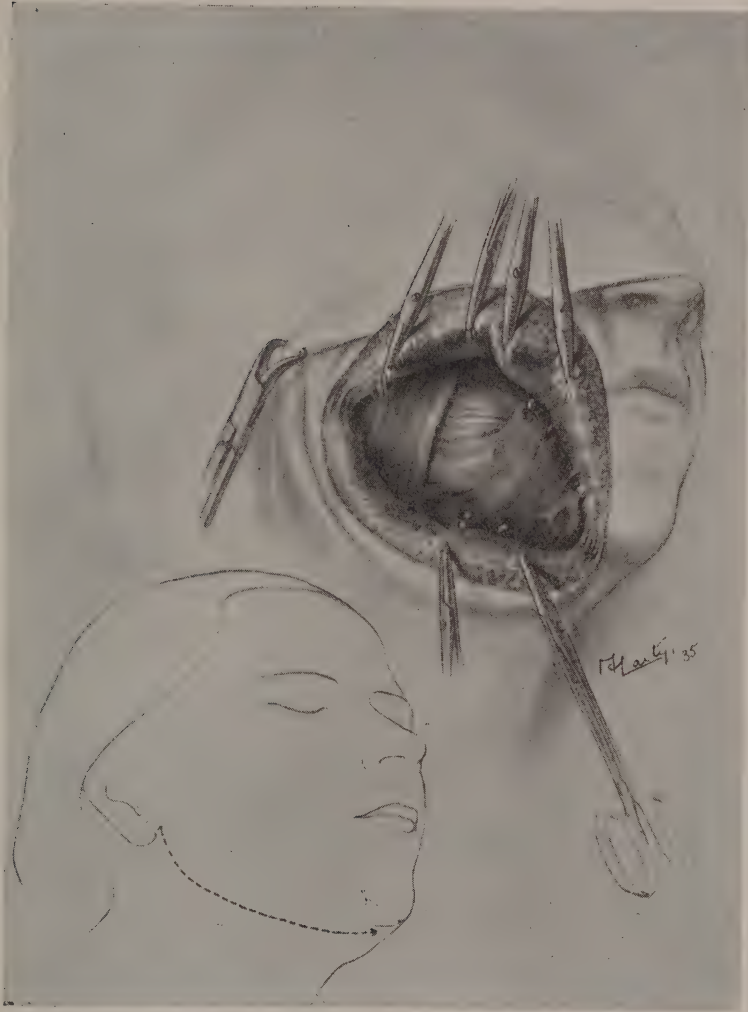


FIG. 1. First stage of operation for carcinoma of the cheek.

excision, and coagulation with a button electrode is carried out. It is best to begin at some distance from the periphery of the tumor and gradually work towards its center. First of all the superficial portions of the growth are dealt with. The coagulated material is re-

moved with a spoon and the diathermy is repeated gradually working deeper and deeper, layer after layer being removed till healthy tissue is encountered.

In the treatment of buccal and pharyngeal carcinoma an artificial route of access is usually unnecessary. The cheek is an exception to this rule, and I shall presently describe what I consider the best method of approach. Resection or removal of portions of the lower jaw is seldom called for. Besides the advantages of a natural mode of access and sterilization of the tissues there are others possessed by diathermy. One of these is the possibility of operating on recurrences a second or even a third time. In cases where the periosteum or bone has become invaded by the tumor there should be no hesitation in applying a strong diathermic current. A sequestrum will form which may take a considerable time to separate, especially if the devitalized bone is a portion of the mandible.

A good view under strong illumination is essential. Where the tumor affects the posterior part of the tongue or the epiglottis adequate exposure of the diseased area may be impossible even if a Davis gag or the suspension apparatus is employed. Any instrument used to depress the tongue is apt to conceal the tumor and it is sometimes necessary to apply traction to the tongue and to carry out part, at any rate, of the operation under indirect vision with the aid of a laryngeal mirror. It is questionable if a growth situated on the posterior pharyngeal wall should ever be treated with diathermy, as osteomyelitis of the cervical vertebræ is likely to follow. When using diathermy in the region of the tonsil and lateral pharyngeal wall the proximity of the large vessels must be borne in mind.

In the case of carcinoma of the mucous membrane of the cheek I found that owing to this structure becoming rapidly thinner as it passes forward from the anterior border of the ascending ramus of the mandible radical diathermy from the inside of the mouth could not be employed without endangering the skin especially in the vicinity of the angle of the mouth. A large lateral opening might result. Carcinoma of the cheek tends to spread over the mucous membrane and later on invades the muscles, but the skin generally remains free except in somewhat advanced cases. In order to meet the anatomical peculiarities of the neighborhood I devised, more than eight years ago, the operation I shall now describe. Where the mucous membrane and possibly the buccinator muscle is involved, but the skin is freely movable, the operation I perform is as follows. After preparation of the skin of the lower face and upper part of the neck



an incision is made, commencing at the level of the lower attachment of the pinna and following the posterior border of the ascending ramus of the mandible and then its lower border nearly to the symphysis. The skin is then dissected upwards so that it is raised from the underlying structures over an area corresponding to, but much greater than the area of mucous membrane to which the tumor is attached. The facial artery and vein are doubly ligatured as they



FIG. 2. Operation for carcinoma of the cheek.

cross the lower border of the mandible and again as high up as possible in the region of the ala of the nose. The portions between the ligatures are excised. This is a most important part of the operation as it prevents immediate and subsequent hemorrhage. A large quantity of ribbon gauze is now packed underneath the flap. The mouth is then widely opened by a gag inserted on the side opposite to the tumor. The tongue is held aside with a spatula and the tumor freely exposed. The growth now can be excised with a diathermy knife from the inside without any fear of damaging the

skin. After removal of the growth the gauze is withdrawn from underneath the skin flap. Through the opening in the buccinator muscle a good view is obtained, and it is easy to make certain that the operation has been sufficiently radical. If in any given case the tumor is seen to invade the upper or lower alveolus the operation can be slightly modified so as to remove this portion of the growth and sterilize the underlying bone. Similarly if the growth extends backwards and is covered by the ascending ramus of the lower jaw good access can be obtained by reflecting the whole or part of the masseter muscle and removing, if necessary, a portion of the underlying bone. The skin flap is now replaced and stitched into position and healing of the skin incision should take place by first intention. In one case I had temporary trouble by leakage of saliva through the skin wound. It is, therefore, important when making the vertical part of the incision not to injure the parotid gland.

Recently I treated a very extensive carcinoma of the cheek which had broken through and involved a small area of skin. In this case I dealt with the tumor by making a transverse incision in the skin which encircled the growth. The skin above and below was elevated from the structures underneath forming an upper and a lower flap. The vessels were secured and ribbon gauze packing inserted. The growth was removed as described in the more simple cases. In order to prevent tension which would have resulted from removal of a somewhat large area of skin a collar incision was made in the neck some distance below the mandible and the skin elevated so as to form a sliding flap. The cheek incision was then brought together without tension. (The skin and the cheek healed by first intention.)

After operation, since the cheek consists mainly of skin, it is necessary to stretch the mouth from time to time with a gag to prevent contraction. The patient can be shown how to do this himself. When healing has taken place there is no longer any tendency to further contraction. It is astonishing that the cheek which eventually forms becomes almost as substantial as the cheek on the non-affected side. Very good results have been obtained from this operation. A gland dissection will, of course, be carried out if necessary.

The after-treatment of any case of diathermy is much the same as when the operation is performed with cold steel. Antiseptic mouth washes should be frequently used. The food must be soft and nourishing. Nasal feeding is sometimes necessary for a time. Secondary hemorrhage will occur occasionally though every means has been employed to prevent it. Sometimes it is very troublesome.

Some months ago one of my surgical colleagues asked me to operate on a case of carcinoma of the tongue which had been treated by radium with temporary improvement. The patient positively refused to have several carious teeth extracted. Prior to diathermy



FIG. 3. End-result in operation for carcinoma of the cheek.

excision I secured the external carotid on the side of the growth. Three weeks after the operation there was a severe hemorrhage followed by another bad hemorrhage a week later, which led me to tie the other external carotid. Notwithstanding this a third hemor-



rhage took place necessitating blood transfusion. Afterwards the patient made a good recovery. These attacks of severe bleeding would probably not have occurred had the teeth been dealt with before operation. In most cases hemorrhage can be controlled by firm pressure. Compression of the common carotid against Chassaignac's tubercle may be employed as a temporary measure. If the hygiene of the mouth is attended to before operation and if in certain cases the main vessel of supply is secured, secondary hemorrhage should be a rare occurrence.

### CARCINOMA OF THE LOWER PHARYNX

When the disease is far advanced the symptoms and signs are so unmistakable that as a rule little difficulty is experienced in diagnosis. In the early stages or even when a tumor of considerable size is present the symptoms may be so slight that they scarcely attract the attention of the patient. The only symptoms complained of may be, for instance, a little discomfort, or a slight pricking sensation perhaps noticed only on swallowing. Pain in the ear may bring the patient to the surgeon. Although some distance from the glottis the tumor may cause laryngitis giving rise to huskiness. Not infrequently the first thing noticed is a swelling in the neck. When there is the slightest suspicion of a malignant growth in this region careful inspection with the laryngoscope must be carried out. Sometimes a little frothy mucus may cover the growth which comes into view after this is cleared away; at other times nothing is visible, or edema is noticed in the neighborhood of the tumor which conceals it from view.

Inspection with a stereoscopic apparatus may be useful in gauging the nature of the surface of the growth and its true size and situation. It is possible in some cases to palpate the tumor. Examination with some form of directoscope may reveal a tumor not visible on indirect inspection, especially if it is situated in the retrocricoid region, or pyriform fossa. X-rays taken when the patient is carrying out a valsalva inflation may help in determining the position and size of the growth.

In considering treatment tumors may be classified according to their situation.

(1) Growths invading the epiglottis and base of the tongue. It is important when both these regions are involved to come to a decision if possible whether the epithelioma started on the epiglottis or tongue. If the greater portion of the tumor is situated in the former region

and seems to have extended forwards the prognosis is much better than if the reverse be the case. Carcinoma of the epiglottis is not as a rule very malignant. A small tumor situated on its anterior or pos-

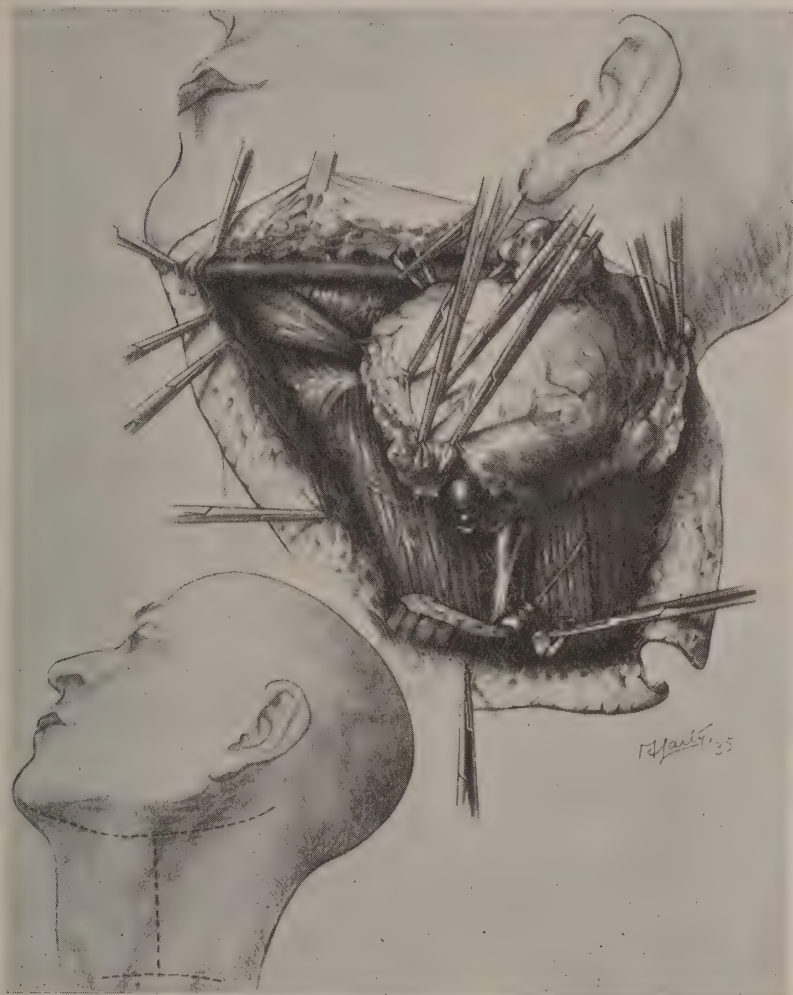


FIG. 4. Operation for malignant gland of the neck.

terior surface or mainly occupying the upper border, even if it spreads on to the aryepiglottic fold or slightly on to the tongue, may in some instances be treated through the mouth by diathermy excision or coagulation.

This may be the method of choice when it is necessary to tax the patient's resources as little as possible. When the tumor is more extensive and the patient is in a fit condition to stand it a lateral pharyngotomy holds out the best hope of complete eradication.

The same statements hold good for a growth situated on the side of the epiglottis or on the aryepiglottic fold. When a tumor appears to have its origin from the region of the posterior part of the tongue



FIG. 5. Operation for malignant gland of the neck and (lower left) specimen after removal.

or vallecula it is doubtful whether surgery should play any part in its treatment. In tumors situated laterally in this neighborhood perhaps originating from the lower part of the tonsil or anterior pillar, I have had a few good results from diathermy. In centrally placed tumors of this part of the tongue, diathermy has not been of much avail. Complete removal may be accomplished by median translingual pharyngotomy, but the growths in this region are usually very malig-



nant, and metastases are generally early and bilateral. Three operations may be necessary to deal with the regions involved and the chances of success are indeed remote. Such tumors should, I think, be treated by radiation.

(2) Carcinoma situated on the posterior part of the aryepiglottic fold or on the arytenoid. This is a rare situation, and these tumors, like those growing from the lateral or posterior wall of the pharynx, are best dealt with by lateral pharyngotomy.

(3) When the pyriform fossa is involved, removal might in a very limited growth be accomplished by means of an approach through the lateral pharynx, but complete laryngopharyngectomy will generally be necessary should such an operation be considered justifiable.

(4) Growths involving the retrocricoid pharynx generally start on the anterior wall of the tube and are almost peculiar to women. If very early they may be dealt with by lateral pharyngotomy with replacement of the excised portion of pharyngeal wall by a specially planned skin flap. When the growths are advanced surgical treatment such as laryngopharyngectomy is not justifiable, and methods of radiation hold out much better hopes.

If the decision is made to remove a cancer from the lower part of the pharynx by surgery the question as to the best manner of approach is not very difficult to answer. Those who have employed the route of approach developed in such a masterly way by Wilfred Trotter will I think admit that his technic is preferable to all others. Trotter's writings are so well known that I shall only mention a few principles which underlie this mode of attack.

1. Careful preoperative attention to any sepsis in the mouth, pharynx or nose.

2. Preliminary tracheotomy removing a disk from the anterior wall of the trachea.

3. In most cases clearing the neck of glands and fascia on the affected side with resection of the jugular vein.

4. Ligaturing any vessels such as the superior thyroid as far as possible from the parent trunk, thus minimizing the risks of secondary hemorrhage.

5. Adequate approach to the pharynx by removing the shield formed by the posterior portion of the thyroid wing and the great corner of the hyoid.

6. Stitching the anterior edge of the sternomastoid and sometimes the cut end of the sectioned digastric over the carotid sheath to the prevertebral muscles in order to prevent the spread of infection.

7. Preparing the prelaryngeal muscles and inferior constrictor as a covering to the suture line in the pharynx.

8. Palpation of the growth if laterally situated through the pharyngeal wall in order to ascertain its size and relationship before incising the pharynx. If at this stage in the dissection the growth is thought to be inoperable the operation need not be carried any further.

9. Packing off with antiseptic gauze all that part of the wound not concerned in the actual removal of the tumor.

10. Up to this stage the operation is a purely anatomical procedure. As soon as the pharynx is opened the upper orifice of the larynx is packed with gauze. In the removal of the tumor the incision must be made so as to excise the growth with an adequate margin of healthy tissue.

11. After removal of the tumor the edges of the wound are accurately approximated with sutures, or if this is not possible, the edges of the wound in the pharynx are stitched to the skin, resulting in a temporary fistula which is later closed.

12. The wound in the neck is filled with boric acid powder.

13. The patient is fed for a time by means of a tube.

More complicated operations have been devised by Trotter when so much of the pharyngeal wall must be removed that its replacement by skin is necessary. In such cases the original skin incisions are made to allow of the cutting of suitable flaps, the position of the base of the flap depending on the portion of pharynx destined for removal. When the disease is so extensive that such procedures are necessary it is questionable whether the humane surgeon will not transfer the patient to the radiotherapist.

I shall not refer to supra-, infra- or trans-hyoid pharyngotomy, or to Trotter's operation of median translingual pharyngotomy which I have already mentioned.

I hesitate even to suggest that Trotter's technic might with advantage be modified in certain cases. I think the form of operation may to some extent depend on what is considered to be adequate clearance of the gland area. Personally I find it very difficult to carry out a satisfactory operation on the cervical glands especially if they are extensively invaded without sacrificing the greater portion of the sternomastoid. Partial section of the muscle does not give a sufficiently good exposure especially of those regions at the extreme lower and upper limits of the neck.

A block dissection might, however, be carried out coupled with the removal of the great cornu of the hyoid and posterior two-thirds of the thyroid lamina. The wound could be kept partially open till

such time as reparative processes had rendered it safe from infection. Then the pharynx could be opened and the tumor dealt with as in the one stage operation. In certain cases after a block dissection only had been carried out on the affected side the tumor might be removed by a transthyroid approach through the non-affected side. In any of these operations should it seem unwise to open the pharynx the tumor may be irradiated by laying needles on the pharyngeal wall as nearly as possible in a position corresponding to that of the tumor. A special measuring instrument might here prove useful. Radium might also be implanted in the tumor if after opening the pharynx it were found to be inoperable or if only part of it could be removed. In the case of a tumor of the lateral wall it would seem to me advisable to remove the necessary amount of thyroid and the great cornu of the hyoid before carrying out the complete and possibly unnecessary gland dissection, as on palpation the tumor might be found to be inoperable.

#### CARCINOMA OF THE NOSE AND NASAL SINUSES

Carcinoma may originate in the nasal chamber or in one of its accessory cavities, generally the antrum or ethmoid. A carcinoma starting in the mouth may spread to the antrum or floor of the nose. It is not uncommon for a basal celled carcinoma (rodent ulcer) arising in the region of the eyelids to invade the adjacent cavities. A tumor of the nasopharynx may spread forwards into the nose. In like manner a neoplasm originating in the region of the cranial cavity may extend downwards through the nasal roof. The classical symptoms of carcinoma of the nose and nasal sinuses are so well known that I need not enumerate them. Unfortunately in some cases symptoms are conspicuous by the absence until perhaps the disease is too far advanced for surgical treatment. In one of my patients the only symptom was frontal headache. On transillumination the antrum was dark, but puncture was refused. He did not present himself again for examination till a year had elapsed and by that time such an extensive carcinoma of the antrum was present that it had passed through the anterior wall and invaded the cheek.

I recently operated on a patient whose only complaint was that he had suffered from watering of one eye for two years. There was a very short history of unilateral nasal obstruction and a swelling of the external nose. I found extensive disease of the antrum with invasion of its anterior wall and roof. The nose and ethmoidal galleries were nearly filled with growth. In another case the patient complained of unilateral nasal obstruction of nine years duration. At



operation the anterior regions of the nose were found to be filled with a collection of cheesy material (rhinitis caseosa), behind which there was a malignant growth situated in the ethmoid and antrum. Neuralgia or anesthesia may be present in some area of skin or mucous membrane supplied by the first or second divisions of the fifth cranial nerve. One of my patients had unilateral loss of sensation over the cutaneous area of distribution of the second division of the trigeminal nerve. The antrum on that side was dark on transillumination. Operation revealed a carcinoma of the antrum which had invaded the floor of the orbit. Another patient complained of pain in the region of the left ala nasi and adjoining cheek. Over this area sensation was diminished. Examination of the nose revealed nothing abnormal. The antrum was clear on transillumination and radiography yielded a negative result. I did not see this patient for six weeks, and to my horror a large firm swelling of the left cheek was present. Exploration showed the antrum to be filled with carcinomatous growth and palliative treatment by radium was the most that could be accomplished. Pain is a fairly common symptom in carcinoma of the region under consideration. Anesthesia is uncommon, but would probably be discovered more frequently if all the cutaneous and mucous membrane areas which might be involved were examined as a matter of routine. It is well known that nerves show great resistance to invasion by malignant disease, but even if not invaded they may be compressed. Signs pointing to implication of the maxillary division of the fifth nerve, or the nasal nerve are of evil omen, as they indicate that the disease is situated in the neighborhood of the upper part of the antrum or nasal cavity.

Sometimes in operating on the antrum by the Caldwell-Luc or other open method a malignant growth is quite unexpectedly discovered. In every case of nasal carcinoma the mouth and nasopharynx should be examined. An x-ray study is always necessary. A great amount of useful information may be obtained from good stereoscopic pictures. As a rule there are no metastases in the neck till late in the disease. In one of my early cases there was a curious sequence of events. I excised the jaw for carcinoma invading the hard palate and antrum. A year and nine months afterwards a carcinoma appeared at the junction of the upper lip and nose. Nearly four years later glands developed on the same side of the neck, but there was no recurrence in the nose or lip. From the growth in the latter region I think the metastases must have resulted.

*Treatment.*—Apart from any consideration of the type of the tumor and its degree of malignancy I think the question of paramount

importance is whether the disease involves the lower or the upper part of the nasal chambers (I use the term to include the sinuses). In some cases both these regions are invaded. Disease low down in the nasal chambers may have originated on the hard palate, or the alveolus, or it may have invaded these structures by downward extension from the nose. As a rule a growth starting in the alveolus especially when teeth are present or when teeth have recently been removed is more serious than one originating on the hard palate. Generally speaking those growths which are situated in the lower part of the nasal chamber are more amenable to treatment. When the floor or the inner wall of the orbit, the regions behind the antrum, or the sphenoidal sinus are involved, treatment is much less likely to be successful. Cancer of the frontal sinus is fortunately uncommon. I have, however, seen a patient where a carcinoma developed in this cavity some time after laryngectomy.

The same conditions which govern operability of a tumor also influence the methods employed in its removal. I have tried all the usual modes of eradicating the disease including formal excision of the upper jaw, and in all but my earliest cases I have made free use of diathermy.

The best route by which to approach most tumors is by removing a portion of the hard palate and alveolus, generally the whole of those structures on the side of the disease. The soft palate is left intact, but I have been driven to take it away when invaded by growth. When the hard palate is not affected a good exposure may be obtained by turning up the cheek as in a Caldwell-Luc operation, and after sufficiently elevating the soft parts removing a large portion or the whole of the anterolateral wall of the antrum. The floor of the orbit, or indeed a large part of the maxilla may be removed by this method of approach.

There are two distinct advantages in taking away the hard palate on the affected side even if not invaded, one being the ease with which the cavity resulting from the operation may be inspected and recurrences early recognized; the other the greater facility and certainty with which postoperative ray treatment can be employed, whether by means of radium needles laid on a mould, teloradium or deep x-rays. In some cases a part or the whole of the alveolus may be retained. The question arises whether all cases should be treated by the palatal route, or whether an external operation is ever called for. I think there are one or two very definite indications for a lateral rhinotomy. If the usual incision is employed every precaution should be taken to ensure that apposition, when the parts are brought to-

gether, is very accurate and the sutures should be inserted close to the skin edges. I find that the scar is scarcely visible if the incision is made with the blade of the knife lying parallel to the cheek so that the skin is cut in a very oblique fashion. The incision can be so made that it is almost completely covered by a spectacle frame. To my mind when the tumor invades the upper part of the nose or the ethmoidal region, where the lower or inner part of the orbital wall appears to be involved, or when the growth has caused an external swelling in the region of the side of the nose, or there is any suspicion that the disease has penetrated the anterolateral wall of the antrum, then an external incision should be employed.

In certain cases the whole operation may be performed through a lateral nasal incision, and I have had some very good results, using this mode of approach but the subsequent view of the cavity after operation is not nearly as good as through a large opening in the roof of the mouth. Had it not been for this opening, I should, at any rate, in two of my patients, have failed to diagnose an easily treatable recurrence. Every case must be considered on its merits and the surgeon should avoid being too stereotyped, or rigid in his methods. A patient, referred to me by one of my colleagues, had been treated by radium with temporary benefit. Examination revealed a carcinoma affecting the whole of the premaxillary part of the upper jaw on both sides. The upper lip was pushed forward by the tumor which was seen to invade the lower part of the nose. As some of the lip had to be removed the case was dealt with by making an incision on each side between the alæ of the nose and the cheek with a continuation downwards through the lip. The whole of the front part of the jaw was taken away and both antral cavities were widely opened. The patient wears a special denture, and he is free from recurrence five years after the operation. He suffers from no inconvenience and there is practically no deformity. I had recently had under my care a patient in whom the ascending process of the superior maxilla, anterior wall of the antrum, and the inner wall and floor of the orbit were invaded by growth, where I thought a lateral nasal incision was indicated. I, therefore, approached the growth which was situated in the antrum and ethmoid first of all from the front and then from below by removing half the hard palate and alveolus. I do not think I could have dealt adequately with the growth by the buccal route only. Whatever the mode or modes of approach diathermy should be used as freely as possible, when dealing with the soft parts. In this way hemorrhage is minimized and dissemination prevented. For instance the mucous membrane over the alveolus, and that covering



the hard palate is divided with a diathermy knife, and with the same instrument the soft palate is severed from the hard. Some parts of the operation are best carried out with a saw or chisel and hammer. Bone can be removed bit by bit preferably after a button electrode has been applied to its surface. The tumor is, as far as possible, desiccated before removal piecemeal with forceps or spoon. Afterwards all cut surfaces are treated with the button electrode, special attention being paid to any suspicious areas. The instrument must be used with care in the region of the cribriform plate, roof of the ethmoidal cells, sphenoidal sinus, and orbital periosteum. Occasionally it may be found necessary to remove the eye. This forms an essential preliminary procedure in those cases of rodent ulcer which invade the orbit, nose and nasal sinuses. Plastic operations may be necessary afterwards when skin has been removed.

Some surgeons recommend preliminary ligation of the external carotid. I have carried it out on a few occasions, but it seems to me unnecessary, especially if diathermy is employed. Probably in all cases of carcinoma in the regions under consideration radiation should be used in combination with electrosurgery, but in my opinion radium or x-rays should never be used alone except in cases too far advanced for operative treatment. Before a surgical attack is made a course of deep x-rays may, with advantage, be administered. After operation the walls of the cavity may be irradiated by radium needles applied on a cast, by which means intermittent treatment can be administered, or telerradiation with a bomb may be employed. I shall leave others who are much better qualified than I to discuss these methods.

#### CARCINOMA OF THE NASO-PHARYNX

My reference will be very brief as I think anyone who has had experience with malignant disease in this region will admit that surgical treatment is not worthy of serious consideration. In the past I have attempted radical operations carried out by various routes with most disappointing results. Some form of radiation holds out the only hope of alleviation or possibly cure.

It has been suggested that such an operation as osteoplastic resection of the maxilla is necessary for the proper implantation of radium, but surely no preliminary operation of this magnitude is necessary. An interesting point in connection with certain malignant tumors of the nasopharynx is the difference of opinion between authorities as to the nature of the growth and the endothelioma of one school must correspond to the carcinoma of another. All those who have made

a study of the subject, and I must specially mention New, Woltman, Trotter and Gardham, are agreed as to the great liability to error in diagnosis, more especially in view of the fact that there may, so often, be no evidence of nasal or nasopharyngeal trouble. Symptoms due to involvement of cranial nerves or even the sympathetic are misinterpreted, attention is focused entirely on pain in the ear or region of the teeth which is purely symptomatic, or glandular swellings in the neck are thought to be primary in origin.

### SOME UNUSUAL CASES

Two male patients successfully treated for carcinoma of the tonsil developed carcinoma of the esophagus, one after seven years, the other after fourteen years.

A male patient with cancer of the left tonsil and anterior pillar treated with diathermy and excision of glands, pronounced inflammatory, developed seven years later cancer of the right margin of the tongue—treated by diathermy together with block dissection.

A male patient had a squamous celled carcinoma of the tongue and at the same time a trabecular celled carcinoma of the rectum.

*Male.*—Operations for cancer of right soft palate uvula and faucial pillars and enlarged cervical glands reported on as inflammatory. Twelve years later developed cancer of right alveolus with secondary carcinomatous glands for which operations were performed. (After the first operation the patient drove an express train for six years and retired at the age limit.)

*Male.*—Operation for squamous celled epithelioma of left tonsil. Five years later developed enlarged hard glands left supraclavicular region. Glands removed and reported on as columnar celled carcinoma, *not* secondary to tonsillar growth. Primary never located.

Female patient treated for carcinoma of the vestibule and anterior region of the right nasal cavity, developed seven years later a hard mass attached to the bone external to the left eyebrow. • Treated successfully by external radiation and still alive nine years and five months since operation.

### END RESULTS

Results are briefly as follows. Nearly all the cases I shall refer to were treated solely by surgical means. It is to be hoped that collaboration between the radiotherapist and the surgeon will lead to improved results in the future.

Carcinoma of the lower pharynx treated by pharyngotomy or laryngopharyngectomy has yielded such disappointing results that I scarcely think, except in selected cases where the disease is relatively benign in type, favorably situated, or very early, that operation is

justifiable. In the majority of cases therefore radiotherapy is indicated.

In assessing five-year results in cases dealt with by diathermy my material is much reduced as patients treated during the last five years are obviously not available for statistics. About eight years ago I commenced to employ radium as a routine treatment except in nasal cases for the primary growth and I kept this treatment up for some years so that my material for the purpose of this report practically excludes all cases treated during the last eight years.

Fifty-one cases of carcinoma of the tonsil and its neighborhood (forty-nine males, two females) were treated by gland excision when necessary and diathermy of the primary growth. Twelve patients passed the five-year period (22 percent).

Thirty-four patients suffering from carcinoma of the tongue and floor of the mouth were treated, many of whom were referred by surgical colleagues and showed advanced disease (thirty-two males, two females). Nine survived for a period of five years (25 percent). In both the tonsil and tongue cases once the five years was passed much longer survival was the rule and the average works out at nearly ten years, some patients surviving for thirteen or fourteen years.

I have dealt with only ten cases of cancer of the cheek by the method I have described (six females, four males). Six are available for the five-year report. One of these died two years after operation and one eight months after but not from cancer. Four are alive, three having passed an eight-year period and one a six-year period. Four more recent cases are all alive and free from recurrence.

Up to five years ago I had the opportunity of tracting but a small number of cases of nasal cancer by diathermy methods. Of ten cases where the nasal cavity and neighboring regions were involved five passed the five-year limit. In seven cases where the hard palate or lower antral region was involved two lived for over five years.

I do not think that the term "cure" should be used. The patient may eventually die of some other complaint when it may be said that malignant disease did not return.

I should like to close my paper by a quotation from that noted astronomer Sir James Jeans, who would have been present, had circumstances permitted, at the inauguration last week of Toronto's great telescope.

The extract is from Sampson Handley's book "The Genesis of Cancer." Sir James makes the following statement: "We know that radium emits energy by the disintegration of certain of its atoms at regular intervals of time. All the atoms in a given lump of radium

are presumably of the same age and in the same condition at the same moment. Yet one of them is taken and the others left. Why?" "We do not know," says Sir James, "nor does there seem any likelihood that we ever shall know." Is the origin of the cancer cell to be, likewise, locked away for all time as a secret? Profoundly we hope that this will not be the case as the key to successful treatment of all cases of cancer may be forthcoming when the cause of the genesis and development of the malignant cell is known.

#### NOMENCLATURE OF CARCINOMA OF EPIDERMIS AND SQUAMOUS MUCOUS MEMBRANES

The nomenclature of carcinomata used in the Bernhard Baron Institute of the London Hospital is essentially descriptive, the growths being named according to the arrangement and morphology of the cells. The carcinomata that arise in epidermis and squamous mucous membranes can in this nomenclature be classified according to their degrees of differentiation towards the normal.

(1) The most differentiated contain *cells with prickle-borders and/or horny cells*.

(2) Less differentiated are growths containing polygonal cells which have no prickle-border but resemble the cells of the malpighian layer in the structure of their nucleus and in the relative amount and sharp differentiation of their cytoplasm; usually there are intercellular clefts that are not crossed by prickles, occasionally these are replaced by an intercellular thickening of the membranes of adjacent cells. Such cells are called "*epidermoid*" because they resemble, but are not identical with, the cells of the malpighian layer.

When growths of types (1) or (2) are composed of cells which resemble those of the basal layer of the epidermis in their small size, relatively scanty cytoplasm and deep staining of their nuclei the qualification "basal celled" is added to the description.

(3) The least differentiated growths are composed of round, polygonal and, occasionally, spindle cells which are not sufficiently differentiated to suggest a definite origin from epidermis. The epithet "*undifferentiated*" is added to the form of these cells to indicate that they are less differentiated than "epidermoid" cells. Such growths appear to correspond to the "transitional carcinomata" of other authors. In these carcinomata of "undifferentiated cells" there is sometimes much infiltration with round cells, and when this is the case the growths appear to correspond to the "lymphoepitheliomata" of certain writers.



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## DISCUSSION

DOUGLAS QUICK, M.B. (Tor.), New York, N. Y. (by invitation): From the standpoint of application of the high-frequency current (call it surgical diathermy, endothermy or what you will) to the treatment of cancer, we must recognize in Mr. Patterson's paper the words of a master. If I make bold to disagree with some features, I do so purely on the premise of fundamental principles involved, and on the clinical experience gained in application of those principles through the years of developing irradiation therapy.

Mr. Patterson employs surgical diathermy in order that he may extend an operation for surgical removal of tissue to wider and safer limits, to parts relatively or totally inaccessible to sharp dissection, with the technic at all times under control and anatomical limits nicely distinguished. I cannot refrain from commenting on his admirable conservatism in avoiding those areas in which control of such a technical method may become doubtful or incomplete. His insistence upon rigid oral hygiene and ligation of vessels where necrosis and secondary hemorrhage is a possibility is equally applicable whether surgical diathermy or irradiation becomes the method of election.

I have occasion to employ surgical diathermy a good deal in my work and, while it is always secondary to irradiation therapy, I can nevertheless support very thoroughly Mr. Patterson's advice on the many technical points, from first hand experience. The manner in which a deeply-fissured infected tumor-bearing area may be sterilized is remarkable; tumor-cell implantation is avoided; and bone, infected or invaded by tumor, can be desiccated and removed quickly without having to wait for the prolonged and painful process of sequestration.

My objection to heat in any form, even the minimum heat of surgical diathermy, is the temporary paralysis of the myriad of small vessels in the zone adjacent to the line of tissue division, and the lack of cellular reaction in this same zone of other than polymorphonuclear leucocytes. The cellular reaction in and around an area of irradiated tissue is predominantly that of plasma cells and lymphocytes. These, as far as we know today, represent the chief cellular defensive mechanism of normal tissues to cancer invasion.

In our various departures from sharp surgical dissection for control of the gross local manifestations of cancer, the efforts of all have been toward extending the effect of the new method or agent as far as possible beyond the sharp line of demarcation which we formerly drew around the malignant growth with a scalpel. Those of us who lean most heavily upon irradiation feel that this safety zone is much wider and adaptable to many more anatomical locations and conditions with x-rays and radium than by any other method.

I am fully cognizant of the general and unfavorable impression which has been created during the early years of radium therapy, and even now by the use of radium in inexperienced hands. We must remember, however, that good radium therapy calls for technical facility, accuracy and experience quite as much as does the most difficult surgical operation. Today we have the advantages of accumulated technical experience, a substantial knowledge of dosage limits as well as a recognition of tumor types best suited to irradiation, and many other secondary factors of influence.

X-ray therapy has gained steadily and rapidly in efficiency, during the past few years. The engineers have given us excellent, dependable and shock-proof equipment up to 200 K. V., and our physicists have standardized it so that we actually know what is being given to the patient. About five years ago Coutard expressed, from his experience, certain principles in x-ray therapy. While I cannot agree with certain radical features of these, I do most fully agree that he reached a high point in x-irradiation. In the interim, many who assume to follow him have so modified his methods for economic reasons that today the best obtainable from x-rays is but seldom achieved. At the present time the best in x-ray therapy is obtained at 200 K. V., using modern shock-proof equipment which affords excellent technical facility, heavy filtration of the rays (2 mm. Cu. or more), long skin-target distance (70-80 cm. or more), slow rate of individual dose delivery (6-10 r. per minute, depending on the size of the field), and treatments daily or semi-daily over several weeks, to make up the total dose.

From my experience in dealing with the groups of cancer assigned to me for discussion today, x-rays and radium represent the backbone of treatment. Every case accepted for treatment receives, first, x-ray therapy as just outlined, and to the limit of reasonable skin tolerance. This x-radiation is given through both sides of the neck and face, to cover the primary growth and the lymph-node bearing areas. Each side is treated daily. All grades of epidermoid carcinomata will, from this radiation, show a marked regression. The totally anaplastic primary growths will regress completely and remain so, if adequate x-ray therapy is carried out; the fully differentiated and intermediate grades may appear to, but this should not be accepted with too much enthusiasm as they are likely to take on renewed growth after a few months. This is more especially true in the oral cavity than in the pharynx, hypopharynx or larynx.

There is something peculiar, and thus far unexplained, in the unusual radio-sensitivity or hypopharyngeal epidermoid carcinoma as compared with a comparable histologic grade in the oral cavity proper. For equivalent grades, those in that surgical no-man's-land, the hypopharynx, respond much more readily than do those in the oral zone. It may perhaps, at least partially, be explained by the amount of lymphoid tissue normally present about the pharyngeal ring.

Biopsy is always done after external irradiation is under way in order that proper histological guidance may be avoidable in determining the ultimate

phase of irradiation. Many of the hypopharyngeal tumors are anaplastic, or relatively so, and hence external therapy is pushed to the limit. Fully differentiated, or the border-line grade II cases, especially if in the oral cavity, receive slightly less x-ray therapy and are finished by radium implantation. For this I prefer radon in 0.3 mm. gold seeds rather than element needles because of the facility of handling, greater technical flexibility, one-stage procedure, and lesser risk of introduction of infection at depth. I have never seen what I could regard as tumor dissemination through radium or radon implantation following proper external irradiation.

Adequate external therapy not only reduces the primary growth and cleans up infection by reason of the reduction in tumor bulk, but it makes implantation safer and reduces the amount of radon needed to complete the necessary dosage intensity. Tissue necrosis by reason of this improved technical combination is very much reduced, and in fact seldom seen, in reasonable cases.

Within the mouth we employ surgical removal of tissue by surgical diathermy, following external irradiation, for access and drainage; for infection or tumor invasion of bone; and for cleaning up of bulky infected fungating tumor tissue in soft parts. In the latter instance it is frequently advantageous to utilize the mass of condemned tissue as a radon holder and remove it by the high-frequency cautery as soon as that usefulness has been accomplished. The cancerous tongue with a background of luetic glossitis and luetic leukoplakia is often best handled in similar fashion, in its entirety or in part, after implantation, because of its excessive fibrosis and decreased capillary blood supply due to the chronic syphilitic changes.

With cancer in the laryngeal box and around the laryngeal cartilages, I have gone through the various phases of total laryngectomy, hemi-laryngectomy, laryngo-fissure and cautery removal, radon by implantation through laryngoscope and through laryngo-fissure, as well as tele-radium with 4-gram pack or bomb. More recent experiences with advanced H. V. x-ray therapy, such as I have outlined, leave us with a series of most encouraging one- to five-year cases free from any evidence of growth. The much talked of cartilage damage is usually the result of inferior technic rather than method. Repetition of series of treatments and excessive daily exposures are bound to produce it. With cartilage invasion by tumor, removal of the cartilage after irradiation, and through laryngo-fissure, is advisable in certain cases to avoid secondary necrosis. If irradiation therapy merits serious consideration in the advanced cases, it is all the more worthy of consideration in the early and favorable cases, and it does not fall short. In my experience, x-ray therapy in intrinsic laryngeal cancer is one of the outstanding advances of the past five years. It is my opinion that laryngectomy for carcinoma of the larynx is now obsolete, and that it will be generally recognized as being such within the next ten years or less.

In most conferences on treatment of epidermoid carcinoma of the upper mucous membrane tract, attention is apt to be focused on the primary growth and little thought given to the equally important problem of cervical metastases. Metastatic involvement of a cervical node at once reduces the patient's ultimate outlook by at least one-half.

I am an advocate of conservative treatment of the lymph-node drainage areas of the neck, rather than routine block dissection, on the theory that up to a certain point at least the nodes perform a conservative and defensive function. As previously stated, we irradiate all necks with x-rays as a preliminary step.



If no nodes are palpable, the case rests at this, but must be kept under routine observation. Over the years we have reduced gradually our groups which we regard operable by complete unilateral dissection. Today it stands at one group—namely, fully differentiated (grade I) epidermoid carcinoma in which the patient is in good general physical condition with primary growth controlled or well on the way to that point, and with the palpable metastatic node or nodes limited to one side of the neck, capsule intact and node movable. The dissection is done under local block and conduction anesthesia and is complete from submental triangle of the opposite side to the acromion of the involved side. If dissection is to be done at all, it should be the most complete and radical possible.

All cases falling outside this very limited histologic and anatomic grouping are treated by surgical exposure and radon implantation. For worthwhile radon implantation, the accuracy attainable only through surgical exposure is imperative for success. Any node which can be accurately implanted by skin puncture is too advanced for the method and had best be treated palliatively by external therapy. Intermediate and totally anaplastic histologic grades, even though technically operable from an anatomic standpoint, are better treated by surgical exposure and implantation rather than by dissection. Even the most radiosensitive growth, metastatic in a lymph node, calls for implantation, in addition to the external therapy, even though it may seem to have disappeared and even though its primary growth has disappeared, simply because we know from experience that the metastatic node is almost invariably more radio-resistant than its primary tumor.

GORDON B. NEW, M.D., Rochester, Minn.: I think we will all agree that during the last decade or two there has been a marked evolution in the treatment of malignancy of the types under discussion today. I think this is well brought out by the treatment of the malignant tumors of the antrum that Mr. Patterson discussed. Years ago these were all taken care of by means of resection, with small operative risk and small mortality, but few of the patients remained well. At the present time we know these patients taken care of with diathermy and radium stay well over a long period of time.

The growth in the upper jaw, of course, rarely metastasizes. Practically all low-grade tumors that extend to the cheek at the same time metastasize as does any other type of malignancy.

Mr. Patterson brought out the classification in relation to the ethmoidal tumors mesially or posteriorly, and that is one of the important things, along with the type of growth, in determining whether the patient is going to get well. One cannot compare growth in the alveolar process with growth higher in the antrum, situated particularly in the region of the ethmoid. That, we know, is a highly malignant region.

I feel that in the low-grade tumors, the low-grade carcinomas, cauterization and diathermy give much better results than can be accomplished by any type of radiation. The surgical removal of the cheek growth gives many possibilities as far as cures in these cases are concerned. I feel, however, that in this group of cases which we take care of by removal of the submaxillary glands, ligating the lingual vessel and taking care of the cheek lesion by means of surgical diathermy, there is a possibility of getting into the submaxillary gland if that

is involved, and going ahead and doing the cheek operation would rather hinder the possibility of a secondary block section.

In the tongue lesions, too, we feel that radiation (and, of course, in the highly malignant tumors) gives the best results. In the low-grade lesions the cautery, excision and diathermy offer a good chance of recovery.

I want to mention one point in regard to lesions just over the base of the tongue and involving the epiglottis. We know the highly malignant ones primarily involve the tongue and a section of the epiglottis. Doing the usual tracheotomy, one can destroy the lesion with surgical diathermy and insert radium seeds directly into the wound.

A month or two ago in such a case I removed the hyoid bone and sequestrum from the inside, showing the extent of the lesion that can be removed in this rather conservative way.

HENRY B. ORTON, M.D., Newark, N. J.: I can only emphasize some of Mr. Patterson's remarks, that surgery, diathermy, and deep x-ray in combination is the procedure of choice in attacking these tumors. The block resection of one or both sides of the neck, with a ligation of the external carotid and a resection of the jugular, is a most important step in eradicating this disease.

Even in some of these carcinomas of the lip in which the carcinoma has been removed from the lower lip, and no palpable glands are encountered in the operation, even if the glands are not palpable before operation, we have found six months or a year following, the glands in the upper part of the neck were involved. Since then we have been removing these carcinomas with a block dissection of the upper part of the neck.

Another important point—in all these procedures, Mr. Patterson has a very, very good exposure. He did not take up the lower pharyngeal carcinoma, but the same point holds good there, namely, a good exposure in attack.

GORDON E. RICHARDS, M.D., Toronto, Ont.: One of the points noticed is that when a radiologist speaks before any audience, and particularly an audience of surgical workers, he is expected to speak in terms of percentages of cases cured at the end of any given period, and when he listens to a paper by a distinguished surgeon, he usually goes away with the impression that he has listened to a series of very beautifully planned surgical operations, the percentage of cures of which were left in doubt.

We would have been delighted if Mr. Patterson had given us a basis upon which comparison could be made between the results being obtained by surgical treatment of these diseases, and those which may be obtained by radiological methods, though there is no thought, wish, or desire to place the two in competition with each other in that sense.

We, as radiologists, feel that the recent advances of the last few years (the last five years especially) have made an enormous contribution to the treatment of malignant diseases of the oral group and of the nose and throat. The claim which we make and upon the basis of which we believe we are entitled to consideration, is the fact that when these methods are successful, they are accompanied by a degree of conservation of tissue which is out of all proportion to anything surgery can do, and unless radical surgery as done, can show at the end of any given period a higher degree of cured patients, then we submit that the radiological methods are superior.

Another thing that we observe is that frequently in discussions of this sort, when statistics are quoted, the best statistics that are produced in the world are quoted, and I decided to be very modest by stating what we are accomplishing in our own work, merely as a basis of comparison.

In carcinoma of the tongue we have had a series of results that were somewhat surprising to me, inasmuch as there has been a tremendous variation in the curability by radium alone, of these lesions, depending upon their situation upon the tongue, and I will show you a slide in a few minutes that illustrates this.

On the base of the tongue we have had the highest percentage of cures, namely, 66 percent; on the lateral margin, 24 percent; on the tip of the tongue, 33 percent; and on the dorsum, none at all. That is by radiological methods only.

In the dorsum of the tongue, in addition to the radiological treatments applied, diathermy and radical surgery have not improved the situation.

In diseases of the tonsil we are in a favorable position of frequently having to deal with transitional carcinoma, which is relatively radio-sensitive, and have reached the general opinion that if these cases were treated prior to the stage of glandular involvement, a high percentage would be cured by external irradiation only in the form of high voltage x-rays or teleradium. However, this does not represent a cross-section of the problem, as we all know.

Taking all cases together, we have been able to obtain 35 percent of cases free of disease at the end of the five-year period. In some of these block resections have been done. If I had time, I would like to make some observations as to the order in which we believe the block resections should be done.

I was sorry that Mr. Patterson did not devote more time to the treatment of carcinoma of the hypopharynx and larynx, because this seems to us, as radiologists, to be one of the fields in which great progress is urgently needed. So far as the radiologist is concerned, as you all know, the radiological treatment was revolutionized by the work of Coutard in Paris, and at the present time all of the work that is done is being done by the method that is known as Coutard's technic, the essential features of which are highly filtered x-rays, at a distance, delivered very slowly, over a long period of time, and worked up to the point of quite severe skin reactions.

In this country the radiologist who has the courage to produce the reactions which are called for, frequently finds himself in trouble, because of the legal consequences of producing reactions which the laity and some members of the medical profession are in danger of labeling as burns, and we dare not produce quite the type of reaction that Coutard does in France. However that may be, it has resulted in a very substantial improvement.

Everyone who is working on this problem has been seeking ways and means of improving these results, or rather, I should say, of improving the technic and those improvements have been sought along two lines: (1) to reduce the skin intensity of irradiation; and (2) to increase the death dose effect.

In our own work we have been running a series of parallel cases, some of which have been treated by Coutard's technic, using high voltage x-rays, and another series has been treated by radium apparatus containing 4 grams of radium, so-called radium bomb. Some months ago we got the idea if we put the bomb in motion and moved it around a circular field, the apex of which was concentrated on the lesion, we might improve the results, and we have had such an apparatus constructed and hope shortly to have it in commission.

Over this week-end, in order to give you some ideas as to whether or not we thought it was going to be successful, I had measurements made and I am giving you this as a preliminary announcement, subject to correction when we have an opportunity of working out the details, which will take some months.

I will show you a photograph of the bomb and also a photograph of the field of irradiation. By putting it in around a circular field, which we can vary from zero (that is, from the center to a six-inch diameter), we can reduce the skin intensity in the ratio of one to seven. The death dose at 5 centimeters is increased 340 percent; at between 5 and 10 centimeters, 540 percent; and at between 10 and 15 centimeters it is increased 500 percent.

Circulating this bomb around a carcinoma in the breast, we can increase the dose which we are able to give between 400 and 600 percent, and it looks as if it would not be necessary to cause a severe skin reaction in order to deliver a very much increased dose in the depths of the tissues.

It is an interesting piece of research which, so far as we know, has not previously been done, and we will hope to spend the next few months investigating the effectiveness of this modification of the bomb. Instead of introducing beams of radiation from different directions, the bomb is in continuous motion, and we introduce a continuous irradiation in the shape of a cone, the apex of which is focused on the disease.

NORMAN PATTERSON, F.R.C.S., London, England (closing): It is a very much more difficult matter when it comes to a question between the surgeon and the radiologist. I am all for the radiologist. I think the radiologists are doing a magnificent work and I hope in the future that although the cases will not, I imagine, be turned over entirely to them, the combination of radiology and surgery will give us very much better results than we have had in the past.

I have not had time to refer to matters elsewhere that I have here in the table. I consider that 35 percent cure of tonsil cancer is extremely good, better than mine, but I hope with the combination of diathermy and radiation to better 35 percent.

The best results I have had so far are with carcinomas of the cheek. I have done ten cases and eight are alive, and one is three years alive, three of them eight years alive, and one six years alive.

I was interested to hear Doctor Richards' remarks on carcinoma of the tongue. In my hands it has been very refractory.

I had no time to deal with the pharynx and laryngopharyngectomy has been far from encouraging. Eventually I think these will be treated largely with radiation.

I see Doctor Quick and Doctor Richards are trying to improve methods of surgery in dealing with the neck, in combination with radiology. It is a good combination, but I would be rather inclined to bank my faith on a well-conducted block dissection rather than on radiation. Few cases of mine have had any permanent benefit from radiation. You apparently get recession for a time and the whole thing comes back.



# THE PROGNOSIS OF MODERATE DEAFNESS IN YOUTH

(VARIATIONS WITH DISEASE, MANAGEMENT AND TREATMENT)

By EDMUND PRINCE FOWLER, M.D.

New York, N. Y.

Since 1922 when I first discussed audiometric measurements before this society, thousands of audiograms have been accumulated with data which shows the trend of hearing over varying periods of time and under varying conditions of disease, management, and treatment.

Deafness is only a symptom but it is usually the chief symptom in ear conditions. Its prognosis depends largely upon the specific lesions causing it, but this is true only in so far as such lesions happen to permanently involve the structures vital to good hearing. Even suppurative otitis media with severe complications is not necessarily followed by deafness, and suppurative otitis media irrespective of the deafness may have a good or a bad prognosis. In cases of long standing, cure may be accompanied not by an improvement in hearing, but by an increase in deafness. Nevertheless much may be learned by studying the average audiogram curves in different disease groups and their variations with time and treatment. The aim of treatment should, of course, be not only to save life and to cure the disease and avoid complications, but to preserve and to improve the hearing. At times unavoidably, but often inexcusably, the hearing is neglected.

Medicine and surgery being, because of so many variables, far from exact sciences, one has become accustomed to rely too much upon isolated cures instead of average results to evaluate the effects of treatment. Average results often damp our enthusiasm, but really are of far more importance than the individual results.

This report is based entirely on clinic patients, who are consequently not quite as favorable subjects for management and treatment as most private patients. The background of inheritance and care and hygiene, etc., is poor compared to the latter. However, clinic cases constitute the bulk of material and the greater problem.

In these cases continuity of intensive treatment was impracticable. It is generally so for obvious reasons, but the usual methods were used, and in all instances supervision was continued over several years with particular attention to general health and head colds and sinus infections.

In no instance, with cessation of suppuration, was there what I consider occlusion of the eustachian tube. Catheterization was not used in these children. It was not indicated. No nasal packs or sprays were used. They were not indicated. Tonsils and adenoids were removed if there was any suspicion of their having a harmful effect on the ear disease or general health. No sinus operations were done. I have not opened a child's antrum in many years. Too much conservatism may have been shown in these matters. In many children the lack of resistance to head infections persists in spite of all operative procedures, and attention to vitamin, clothing, hygiene and other health measures.

It is by no means an unmixed blessing in the very young to remove the tonsils unless they are chronically diseased (not simply hypertrophied). These structures like other lymphoid masses serve a useful purpose. Their autogenous products are more efficient than any manufactured vaccines. I am of the opinion that unless adenoids (with or without the tonsils) are removed soon after chronicity or recurrence is threatened, and before the child's ninth year, the procedure does not contribute much on the average to immunity from otitis media.

It is often a nice decision as to whether one is to do more harm than good in removing tonsils. Some most persistent suppurative ears, and severely deafened ears immediately follow tonsil and adenoid operations. More tonsils are removed in clinics than in private practice. This may be advisable but there is no general rule to follow. That indefinable thing called good judgment must ever be the imperfect but best guide.

At this time I shall limit my discussion to four disease groups in the order of their destructive propensities toward ear health and hearing. The worse ear in every case at sometime had harbored chronic, recurrent, healed or past marked suppurative otitis media. The better ears, almost without exception, also had been the seat of like lesions. The term "past masked" was coined to cover those cases which, though giving no history of otitis, showed clinical and otoscopic evidence of otitis media in the past, this fact being covered up (or masked) by time, other diseases, or carelessness. The healed cases represent those who gave a definite past history of suppuration. These two groups cover what is usually called otitis media catarrhalis chronica.

It is practically impossible to select absolutely clear-cut cases because there are always many factors in even the simplest clinical entity, but I have excluded three of the complications which would

have greatly confused the picture, namely, otosclerosis, nerve deafness, and mastoidectomy. The first, by the absence of typical symptoms and the ages of the patients; the second, by including only those with normal bone conduction; and the third, by placing the mastoid operations in a separate group. All of these will be reported upon later. These eliminations account for the comparatively small number of cases under each group, plus the tendency of patients to drift away from the clinic, no matter what the results of treatment may have been.

In order to know what we are talking about it is desirable to qualify the degree of deafness. My habit is to grade deafness as follows:

Not over 20 S. U. loss indicates slight deafness.

Over 20 not over 40 S. U. loss indicates moderate deafness.

Over 40 not over 60 S. U. loss indicates moderately severe deafness.

Over 60 not over 80 S. U. loss indicates severe deafness.

Over 80 S. U. loss indicates very severe deafness.

No hearing, for very loud sounds, indicates total deafness.

If desired the word "marked" may be substituted for "severe."

These cases were selected because they were in the moderately deafened group of children who returned for treatment or check-up at least once yearly for several years. The cases present a fair sample. The moderately deafened constitute the largest class of children handicapped by deafness.

## SUMMARIES OF AVERAGE CHANGES BETWEEN TESTS

### CHRONIC SUPPURATIVE OTITIS MEDIA, TWENTY-TWO CASES

The difference in the level between the better and worse ears is greater than in any of the other three groups.

In both the better and the worse ears 8,192 is the least depressed tone and 512 the most depressed tone.

The curve form changed but little with time.

The spread of losses, as between frequencies, is greatest at the first test, especially in the better ears.

The curves became flatter as time went on.

At the second test there is no improvement on the average in either the better or the worse ears.

At the third test there is no improvement on the average in the better ears, and a loss on the average in the worse ears.

At the fourth test there is a definite loss in both the better and the worse ears. Subsequently seven of these cases improved considerably, but the number was too small to warrant plotting.

### RECURRENT SUPPURATIVE OTITIS MEDIA, TWENTY-THREE CASES

The difference in the level between the better and worse is less than in the chronic suppurative ears, but more than in the non-discharging ears.

In both the better and the worse ears, 512 is the most depressed tone, and 2,048 the least depressed.

The curve form changed considerably with time. In the better and worse ears it became flatter.

At the second test there is a little improvement in the better, and a marked improvement, on the average, in the worse ears.

At the third test there is little change in the better ears and slight loss in the worse ears.

At the fourth test the better ears show marked improvement, the worse ears lost hearing.

#### HEALED SUPPURATIVE OTITIS MEDIA, TWENTY-SIX CASES

The difference in the level between the better and the worse ears is less than in the suppurative ears, but more than in the past masked ears.

In both the better and the worse ears 512 is the most depressed tone.

In both the better and the worse ears, 8,192 with 128 are the least depressed tones.

At the second test there is no change in the better ears.

At the second test there is a slight improvement in the worse ears.

At the third test there is a tendency to go down in the better ears.

At the third test there is a tendency to remain stationary in the worse ears.

In the fourth test there is a loss in middle and higher frequencies in both the better and worse ears.

Otherwise the tendency is to remain stationary in both.

#### PAST MASKED SUPPURATIVE OTITIS MEDIA, TWENTY-FOUR CASES

In both the better and the worse ears 512 was the most depressed tone.

In the better ears 8,192 is the least depressed.

In the worse ears 128 and 2,048 are the least depressed tones.

At the second test there was an improvement in hearing in both the better and worse ears.

At the third test the improvement was maintained in both the better and worse ears, although both ears showed a tendency to loss in the higher frequencies.

#### SUMMARY OF OBSERVATIONS ON THE AVERAGE CHANGES IN THE HEARING FROM YEAR TO YEAR IN FOUR GROUPS OF OTITIS MEDIA (190 EARS)

The better ears are on the average distinctly above the worse ears (five to twenty-eight db).

The difference in levels between the better and worse ears was greatest in otitis media, suppurative chronic, and diminished on the average in the recurrent and non-discharging groups.

The average audiogram curve form, changed but little with time, in the worse ear.

In some instances with time the better ear became the worse and vice versa.

Spread of losses as between frequencies was greatest at the first test. In other words, the curves became flatter as time went on, the low tones on the average going up, and the high tones going down in all the worse ears and markedly in the better healed ears.

In all the better and in all the worse ears 512 was the most depressed tone.

Five hundred twelve was most depressed in the recurrent and chronic suppurations.

Eight thousand, one hundred ninety-two (with 128 in healed and past masked worse ears) was the least depressed tone.



At the second test in all the worse ears except the otitis media, suppurative chronic, and in the better healed ears there was improvement in the hearing. This can, therefore, hardly be due entirely to the practice factor. Most of the ears with the exception of the chronic suppurative ears had been tested before coming to the clinic. I have noted that the lower the graph the less the practice factor enters. It may be because on the average these deafer ears are not so much disturbed at the first test by outside masking sounds, as are the better hearing ears.

## SUMMARY OF CLINICAL HISTORIES (AGES FIVE TO EIGHTEEN)

<i>Number of cases</i>	<i>Age limits and average age at first test</i>	<i>Positive family history of deafness</i>	<i>T. and A. ops.</i>	<i>Diagnosis, better ears</i>	<i>Diagnosis, worse ears</i>	<i>Subsequent history</i>
22	(6 to 16) 9	7	16 Out 1 Hyp. 5 Norm.	11 OMSC. 7 Rec. 1 Poly. 8 Hld.	OMSCh.	With the possible exception of three, none appeared healed permanently. Fifty percent continued chronic. Seven at the fifth and sixth tests showed better hearing. Others gradually lost hearing.
23	(5 to 18) 10½	10	17 Out 3 Hyp. 3 Norm.	All Rec. but one	OMSRec.	In 75 percent the frequency of recurrence declined. Better ears gained. Worse ears gained at first, then lost.
26	(6 to 18) 9½	3	22 Out 1 Hyp. 3 Norm.	All Hld. or pst. mskd.	OMSHld.	None developed ac. otitis. One ch. otitis after pneumonia. All picked up in school tests. Better and worse ears lost in middle and higher frequencies.
24	(6 to 16) 10½	12	20 Out 1 Hyp. 3 Norm.	All pst. mskd. but one	OMS.— pst. mskd.	None developed ac. otitis. All picked up in school tests. At second test both better and worse ears gained and maintained this gain over later tests.

With the exception of the healed group, the positive family histories increased indirectly with definite histories of previous chronicity. This would imply that in the healed group at least, there was little hereditary influence.

On the average the younger the patient when first seen and the more faithful in treatment, the less the recurrences and exacerbations, and the less permanent or progressive the losses in hearing.

In the better ears on the average the recurrent and past masked eventually improved.

In the better ears on the average the healed and the chronic showed improvement.

In the chronic, better and worse ears, there was a definite tendency downward.

In the recurrent better ears there was an improvement at the fourth test, but in the worse ears after the gain at second test, a loss.

In the healed better ears there was a definite tendency downward.

In the healed worse ears there was a definite tendency (after a primary improvement) to remain stationary.

In all past masked ears there was a definite tendency to improve and retain this improvement.

The better healed ears show the greatest losses with time.

Whereas these results show the average tendencies, there were many variations, and on the master graphs where each case is numbered many cases show marked improvement, although the average show loss of hearing.

Is it possible to single out a few factors which appear to make for a favorable prognosis? I believe it is. Of primary importance is the early detection of ear disease. In the chronic and recurrent groups loss in hearing was proportional to the chronicity and severity of the suppuration. Fifty percent did not heal even temporarily under treatment; these were the deafened ears of largest duration. There were no reliable histories of the severity in the healed ears. The freedom from acute otitis in the healed and past masked ears is significant. Management and treatment did help these cases.

Pain and distress compel attention. Slight and moderate deafness does not. Pain or distress occurs almost wholly in acute otitis. Either is usually transient, soon forgotten, and of less importance from the prognostic viewpoint than deafness, but slight deafness being usually unsensed, they loom large as danger signals.

Be on the lookout for even slight deafness. This is the danger signal so constantly present and so constantly ignored. It is a warning of danger ahead and of danger behind. Repeated, it is a sign of impending progressive deafness, no matter how slight its beginning. It is the healed ear which more often is allowed to drift. Even the slightly deafened healed ear often goes on to severe and progressive deafness.

I sometimes think that infants should be conditioned to a standard sound of say, 10 or 15 db. above minimum audibility, preferably a low tone (about 256). They certainly are automatically and easily conditioned to sounds they learn to associate with feeding preparations. Why not have the mother or nurse use an acumeter or some such sound or a faint whisper at twenty feet, at first before each feeding, often enough to maintain the association, and to make

it possible to detect any change in the baby's hearing acuity. At twenty feet the above sounds reach the ear at about 15 db. above average normal minimum audibility in a fairly quiet room. Any other sound near threshold of normal audibility may be used. Why not try it out? It is not so difficult, or so foolish as it seems at first thought.

These findings indicate that the continuation or recurrence of suppuration is definitely injurious in spite of the fact that while the ear is discharging the hearing may appear better. Many ears healed long ago (whether diagnosed or not) have had repeated inflammatory episodes, and show little further improvement after a primary response to treatment.

The most surprising losses are in the tones above 512 in the healed ears. This is no doubt partly due to the fact that those who returned for the third and fourth tests were mainly those who noted increasing deafness. The others had no urge to return. This urge factor is operative only in ears with compelling symptoms. The healed ears are in reality subject to recurrent inflammations, coincident with and often caused by nose and sinus infections and exacerbations. These influences may be traced throughout all progressive deafness due to otitis media in any form, and they were so treated.

In one sense all these children have been neglected children. Hardly without exception they were not brought to the clinic until forced by excessive symptoms, or school hearing surveys.

I am sure that had they been seen earlier the story would have been quite different. It has been so in most instances where early examination and proper care was instituted.

By proper care I mean not only the supervision of recovery from pain and discharging ears, but real treatment and management during and after the pain and inflammation have ceased. Also instructions in the avoidance of recurrences.

I have shown figures which tell the story of the average child with suppurative ear disease (apparent or masked), and particularly the story of potential and increasing deafness with time, and lack of early and proper treatment. The figures show that this clinic treatment and management is often not satisfactory. The results on the average are not flattering and yet the individual audiograms show that much can be done to prevent deafness. The prognosis on the average is favorable with youth and early treatment. The prognosis is not favorable even in children unless there is early care and treatment.

## DISCUSSION

MAX A. GOLDSTEIN, M.D., St. Louis, Mo.: Mr. President, there is much of careful observation, rational conservatism, and common sense in this paper. The reference to the treatment and unnecessary removal of tonsils in this particular type of observation is a pertinent one, and it comes home to me rather seriously that at the Institute, where even a more serious form of hearing defect, that of congenital deafness, is involved, in my inspection of several hundred cases that come to us from various parts of the twenty to twenty-five different states, congenitally deaf children almost all have had their tonsils removed, and not only the tonsils but in many instances parts of the uvula, and so forth, which may interfere with good speech and proper physiological function of these parts that are so necessary to speech.

It is rather a sad thing to contemplate that it is still customary to remove ordinary, hypertrophied tonsils in congenitally deaf children, with the thought that it may improve the hearing.

A deaf prevention clinic like that conducted by Doctor Fowler and elsewhere, is one of the newer developments from an otological point of view, in the consideration of this question of the hard-of-hearing child, but we haven't gone far enough. It still has its limitations.

After the otologist has delivered his opinion as to the character of hearing defect in the many children who come under his observations through tests of the group on the audiometer, and after they are sent to him for further test, inspection, and advice, it still remains for the school authorities of that district to help carry it out. If that child shows, as Doctor Fowler did in his summary table, various types and degrees of defective hearing, some definite measure should be undertaken by the school authorities to cope with this question. It is being agitated; it is in the air; but we haven't quite reached a good practical program for the development in most of our communities, of this thing.

The hint that otosclerosis in Fowler's classification should be considered from an age point of view has its limitations.

About ten years ago Alexander reported a case of clinically diagnosed otosclerosis in a girl of twelve, in whom he had seen the temporal bones a few years later and substantiated the diagnosis under the microscope.

A few years ago Stacy Guild, at Baltimore, at the Otological Laboratory of Johns Hopkins, found accidentally, without having a record of the clinical character of the case, a typical otosclerosis in a child of two.

I have now under observation, and it is quite a puzzling case, a child who was brought to us three years ago, apparently a congenitally deaf child. She was then three years old. When digging into the family history of this case, we found five generations straight in a line of otosclerotics, about nineteen members in the family. I had expected to present that to the Otological Society this year, but we weren't quite ready. It is an exhaustive study of five generations.

Albert Gray made one of three, and we would like to contribute one with two generations additional, in this picture.

Now what is this child? Is this child a congenitally deaf child, or has the otosclerotic process developed as an hereditary thing or at birth? If you can have otosclerosis in a child of twelve and a baby of two, why not heredity and otosclerosis at birth?

It is a possibility. It ought to be thought of. There is always a passing smile when we speak of *otitis media catarrhalis chronica* in 1935. I agree with Doctor



Fowler. As he says, "The loss in hearing, in chronic and recurrent groups, is proportional to the chronicity and severity of the suppuration, and not only is it dependent on the severity of the suppuration but on a continued suppuration of rather severe type going on for a period of years." There are so many of those, and the constant, persistent irritation produced by the suppuration itself, and by the mechanical manipulation of treatment, so severe that some of these cases with hereditary tendencies to otosclerosis (and we have admitted otosclerosis is about the only thing in otology that is hereditary) may be provoked to active stimulation of the otosclerotic process.

I like this emphasis on the ignoring of the slight deafness in acute cases, as a danger signal. I think that is a wise and timely observation.

For one moment to revert to the otosclerotic possibilities, we know that almost any constitutional upheaval will begin an otosclerotic process, and in every pregnant woman, with the birth of each child (with the mother having an otosclerotic background), there is an increase in deafness.

Why not follow up Fowler's suggestion? With a constant presence of chronic suppuration and an irritation by mechanical interference, why not consider that as a possible cause for the development of an otosclerosis, or the stimulation of an otosclerotic process?

In conclusion, may I say that perhaps the most timely suggestion that is made in this paper is the fact that the otologist not only shoulders a responsibility in connection with every hard-of-hearing child, under the conditions of the clinical character and picture of the case, but also this follow-up problem is an important one, and it is up to the otologist to help in this follow-up and to investigate, and to advise in the future conditions of the case.

GORDON BERRY, M.D., Worcester, Mass.: From year to year we find ourselves looking to Doctor Fowler for new ideas in the study and care of our hard-of-hearing children. This year, and perhaps for the first time, we have been shown progressive changes in hearing as it concerns a group rather than an individual. How much do we really know about progressive changes in large groups? We know individual trends but what does the average show?

It has been my privilege to examine some of the material developed by Dr. Ruth Guilder and Miss Louise Hopkins in their careful researches on the deaf pupils at the Clarke School for the Deaf, at Northampton, Mass. In a paper before the Eastern Section meeting of our Society last January, we reported that all these children had some hearing, and that most had a nerve form of deafness where the average graph showed a descending curve.

Periodic audiometric tests in these children have been continued over nearly four years now and their analysis is giving valuable data. We suspect (1) that there is a more rapid recession in hearing in their earlier years at the school, (2) that this recession is intermittent rather than continuous, and (3) that it is synchronous with recurring colds. If true, these are significant findings from both the medical and the educational points of view. I am hoping these research workers will soon have enough data to substantiate this suspicion.

In Worcester we have been carrying forward phonoaudiometric tests and otological examinations in the public schools for three years. In 21,000 tests, Dr. Harry B. Goodspeed has found an appreciable hearing loss in 5 percent. Secondary tests, after the institution of early therapeutic aid, have shown definite gains in the hearing of many. But, on the other hand, a few have shown a

gain without having had otological aid in the interim. This confuses the issue. The nerve deafness cases do not offer so favorable a prognosis. Their hearing acuity has remained approximately stationary during this period.

We learn with interest that so many of Doctor Fowler's children showed a gain in hearing. He does not catheterize. No nasal packs or sprays are used. He does no operative sinus work. He cautions against wholesale tonsil surgery. Many of us find these sound doctrines. He does an adeno-tonsillectomy when indicated. Care in general hygiene is observed. Recurring colds and sinus infections are given routine treatment.

Are politizerizations used? How are suppurative ears controlled? If the adenoids alone are removed, is the aftergrowth of pharyngeal and nasopharyngeal lymphoid tissue more, or less, active than after the removal of both the tonsils and the adenoids? Such are questions that naturally arise, but which the shortness of his time did not permit elaborating.

As to whether tonsils furnish autogenous vaccines, in my experience, a diseased tonsil may serve as an immunizing agent in a child, but it is very doubtful if it so serves in an adult.

It is significant that the 512 or conversational range is the one suffering most and the 8,192 or high range shows the least loss. Of special importance is Doctor Fowler's assertion that early detection of the difficulty gave the best results. This reiterates the claim that routine school testing is necessary. Only so can these cases be discovered early. Over and over again those who do these group tests discover fairly advanced forms that neither teacher nor parent nor child was aware of. Early detection and conscientious care will, in my opinion, lessen or eliminate a very considerable proportion of our advanced cases in adult life.

J. W. JERVEY, M.D., Greenville, S. C.: Mr. President and Members: I would be the last one to question anything that Doctor Fowler had done, but I am a little sorry that he has grouped all of his cases of moderate deafness under one head, and has given us a general summary of averages, so to speak, as to the outcome of the cases.

What I want to get at is that there are so many different causes of deafness, from slight, moderate deafness, to severe and entire loss of hearing, that naturally, grouping all of these cases together, the average will give us a wrong impression as to what might be done in certain individual classes of cases.

Perhaps many of you will remember that a few years ago Professor Hajek, in Vienna, at the celebration of his retirement, at a banquet made this statement: "In my clinical lifetime surgery has practically reached its perfection. Medicine henceforth must look to the study of biochemistry and nutrition."

Now, the point that I wish to emphasize is that in many of these cases of even more or less advanced type, the cause which might be found in the study of biochemistry and of nutrition must be very seriously taken into account in order to arrive at a reasonable prognosis, and I firmly believe that with the further study of that group of cases, which undoubtedly comprises perhaps the largest group of cases of deafness, we will see results which heretofore we have never been able to attain, and I hope that more time and more attention and more study is going to be turned to the consideration of nutrition and biochemistry in relieving these conditions, and I think our statistics and our figures will be vastly improved.

EDMUND PRINCE FOWLER, M.D., New York, N. Y. (closing): I think if Doctor Jervey will read the paper, he will see just what I avoided doing was to group all the cases together. We divided them into groups on purpose not to group all the cases together, but we put them into four clinical groups.

Of course, there are other clinical groups, many more besides this, but that is enough for one meal. There are four of these and we will try to work out the others later.

Regarding otosclerosis, Doctor Goldstein brought up the point that these children may have otosclerosis. That is true. We find one in twenty people in general at autopsy in a large hospital shows otosclerosis, whether they show deafness or not. Many do not show deafness because the otosclerosis does not involve the stapes.

We have to exclude them in some way. I don't know any other way to exclude them except by not having a family history, possibly, which is rather indefinite, because we all inherit our parents' and grandparents' looks, outside and inside. We have the same tendency to certain diseases that they had, irrespective of whether it is otosclerosis or not. It may not be traced in a definite genetic way, but certain families we have seen in our practice tend to have certain things. It may be that the infectious element comes in because they live together, but also the anatomical background is there, otherwise they wouldn't have the same faces as their fathers and mothers.

Now, it is difficult in clinics to get over any complicated system of dietetics and biochemistry. It is hard to get any treatment at all. What is the good of a child's coming in once a week for politzerization? Even in private practice, if you have them come in once or twice a week for catheterization or politzerization, what good is that from a really basic standpoint? It is only a drop in the bucket. They have to do something besides that to get over these things and they have got to do it at home.

I have told you before what our treatment has been, various grades, and so forth, which they use at home to help themselves.

# ANATOMICAL ANOMALIES OF IMPORTANCE TO THE OTOLARYNGOLOGIST

By OSCAR V. BATSON, M.A., M.D.\*

Philadelphia, Pa.

The subject of anomalies is always intriguing. Perhaps it is their seeming mysterious character that appeals to us. We have the feeling that here is something beyond our power of comprehension. An anomaly should, however, challenge us to interpret the structure which has been defined as "contrary to rule." I have always been impressed with the extremely normal appearance of anomalous structures as they lie in the body. The whole organism is adapted to them. We may recall that it was this normal appearance, coupled with the small number of specimens available to him, that led Vesalius to describe as normal an ossicle in the foot, the *os Vesalii*, which we now know to be an anomaly.

Since the otolaryngologist looks upon each skull in a collection as an unusual specimen, especially in reference to the paranasal sinuses, to even enumerate all of the observed anomalies of interest would be impossible. To narrow the field and to allow for a more detailed consideration, I have chosen to describe some of the vagaries of the arterial system that are found in the head and neck. These are of considerable practical importance. Some of these anomalies are due to aberrations of development and as such their presence may not be forecast. When encountered, however, it may be possible to interpret their presence.

One of the most unusual of such anomalies is the case of the doubling of the aorta, such as described by Homer Blincoe, *et al.* (1932). In this specimen, which I have had the opportunity to examine, the aorta splits to enclose the trachea and esophagus and recombines posteriorly. The genesis of this condition with a review of the older literature will be reported by Blincoe. This is a very rare anomaly, but not unique. While it probably would not occlude the trachea and esophagus, it would give peculiar radiographic and endoscopic findings.

There is an anomaly, however, due to peculiarities of transformation of the aortic arches, which is of decided interest to the

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\* By invitation.



otolaryngologist. It occasionally happens\* that the right subclavian artery does not spring from the innominate artery, but comes from the descending part of the thoracic aorta. Under these circumstances, due to the disappearance of the proximal portion of the embryonic right aortic arch, the right recurrent laryngeal nerve is not displaced inferiorly by the right subclavian artery. It comes directly from the main stem of the vagus nerve and courses directly over to the larynx posterior to the superior thyroid vessels. Considering that the upper thyroid pole may not be handled with as great care as the tracheoesophageal angle, it is obvious that this "recurrent" laryngeal nerve may be damaged in the thyroid operation.

There are a number of other anomalies which, like the two major ones just noted, would fall under the head of irregularities of transformation of the embryologic vascular pattern. To be included here would be the origin of the external maxillary artery from the internal maxillary artery and its consequent relation to the parotid gland instead of its usual relation to the submaxillary gland.

Mention should also be made of such balancing arrangements as are commonly seen. The transverse scapular and the transverse cervical arteries are of reciprocal size. This is true also of the anterior and posterior ethmoidal arteries. When the surgeon finds a small anterior ethmoidal artery in the radical frontoethmoidal sinus operation, he should expect this to be compensated by a large posterior ethmoidal artery.

There are a number of anomalies of the arterial system that have been described as anomalies of position, which upon more complete study may be shown to be fundamentally anomalies of size, *i.e.*, length and diameter. One of the most interesting is to be found in the anomalous innominate artery. Dr. Chevalier Jackson (1934) has called attention to the relative safety for surgical approach to the trachea, of a midline triangular area lying medial to the two sternocleido mastoid muscles. So safe is this triangular area for tracheotomy that Doctor Jackson has labeled it "safety." However, in the specimen here described the innominate artery appears in the neck on the left side of the trachea, crosses the trachea in the triangle of safety to divide near the right border of the trachea into the left subclavian artery and the left common carotid artery (Fig. 1).

\* The frequency of occurrence of this condition depends upon the racial background of the material examined. Adachi (1928) has furnished some tabulations on this point. In an English series it was seen five times in 500 cases. Adachi saw but one case in 516 cadavera (Japanese) examined. In those American laboratories reporting the findings in the negro, the incidence is much higher. At Pennsylvania we see one or two a year.

This indeed would seem to be an anomaly of position and, of course, it is. However, an examination of the other large vessels shows first of all that the first part of the aorta is markedly dilated and lengthened. It presents the typical picture of the aorta dilated by hypertension and arteriosclerosis. This lengthening of the first part of the aorta caused some torsion of the heart and at the same time pushed the point of origin of all the vessels to a greater distance from the heart. Here then we have an apparent anomaly—it has

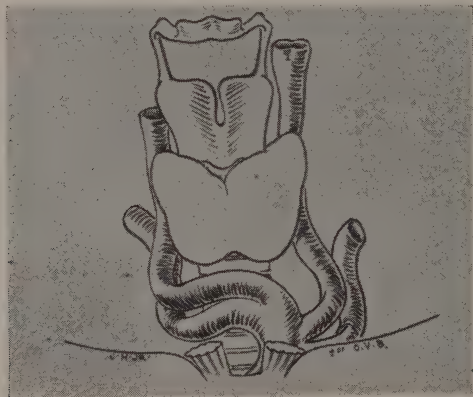


FIG. 1. Innominate artery with its branches lying in front of the trachea in superior mediastinal area. Adult, black male.

been described as such,\* which, on analysis, is seen to be due to a pathologic change in the vessel. The anomaly of position is a result of the pathologic change. The importance of this vessel to the operation of tracheotomy is at once apparent. Palpation of the triangle of safety would prevent surprise. This condition is seen at least once every other year at the laboratory of the Graduate School of Medicine. This means that it occurs about once in two hundred cases in our material. It seems to be more common in the negro.

Another vessel sometime seen apparently out of its normal course, and which obstructs a surgical approach, is the inferior thyroid artery. The retropharyngeal space and posterior mediastinum may be approached low in the neck. This site has been used for operation

\* Symington (1887) described this condition as normal for the child. His report seems to be the fundamental basis for the acceptance of this relation as normal by Merkel (1899) and others. Personally I have never found the innominate artery about the sternum in the newborn. I have not had the material available to investigate this point in children. In the adult specimens that I have seen, this condition is always associated with gross pathologic changes in the arteries. The case illustrated by Adachi (1928, p. 27) appears to fall into this class of pathologic vessels. The figure of the case reported by Jaensch (1922) undoubtedly represents a case of arteriosclerosis. This is also the opinion of Adachi (1928, p. 43) concerning the case of Jaensch.

on the cervical esophagus. Normally an incision through the skin and superficial fascia allows an approach to the area. The great neck vessels are retracted forward. In two or three out of each one hundred of our specimens, the inferior thyroid bends cranialward as an inverted "U" (Fig. 2). When seen through the operative field, the vessel at first causes confusion. It is so large and so far from the usual course of a vessel. Complete dissection, as is



FIG. 2. Inferior thyroid artery extending upward in an inverted "U."

possible in an anatomical laboratory, shows this to be a dilated, elongated, inferior thyroid artery bending upward and into the areolar tissue of the retropharyngeal space. It springs, by way of a rather tortuous thyroid axis, from a twisted and dilated subclavian artery showing the character of hypertensive arteriosclerotic vascular disease. This vessel seems to be more commonly displaced on the right side of the body.

The common carotid arteries and their branches also show anomalies due to increase in diameter and length. This may vary from a slight sinuosity to the formation of a complete loop. The complete loop, when present, is seen in the course of the internal carotid artery. The feature of interest to the otolaryngologist is the appearance of these vessels when exposed by the oral approach. This elongation of the arterial tree no doubt explains many, if not most, of the "anomalous" vessels seen in adults during tonsillectomy. Oc-

casionally, such vessels are seen in children and they may be due to a genuine congenital anomaly. I am inclined to feel that most of the adult cases are due to vascular disease. The purported tortuous carotid artery of the seal has been mentioned to provide phylogenetic reference for this condition. Carmel (1928) has shown that this observation of Chauveau has not been confirmed by other authors\* and he himself could not confirm it on twelve dissections of the seal. In man the artery need not make a complete loop to obtrude into the retrotonsillar area (Fig. 3).

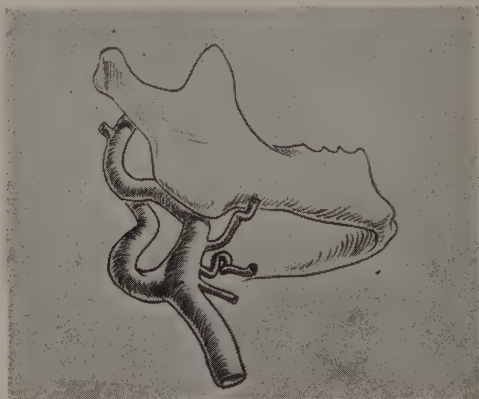


FIG. 3. Tortuous arteries at the angle of the jaw in a cadaver with extensive dilation and tortuosity of all vessels. Adult, black male.

The next vascular peculiarity to be examined is the carotid artery in its course through the bony carotid canal. Here a dilation with some increase in length causes an erosion of the carotid canal. This is significant because of a possible pitfall in diagnosis. With the recent interest in erosion of the apex of the petrous portion of the temporal bone through infection, many x-ray examinations are being made of this area. In the vertico-occipital view of this region, the erosion caused by the dilating carotid may be so closely related as to confuse the uninitiated. The mentovertical view differentiates

\* The first French editions of Chauveau (1857) and the second edition of Chauveau and Arlong (1873) were not available to Doctor Carmel. I have examined these and find no reference to the miraculous artery of the seal. Fleming (1871) introduced this statement into the first English edition of Chauveau and it has appeared in subsequent editions.

Smith (1902) in reporting his case refers to the tortuosity of all the vessels. Fisher (1915) and Schaeffer (1921) inclined to the phylogenetic view. The more complete the exposure of all of the great vessels at one time, the more obvious the generalized nature of the condition became. Adachi (1928, p. 98) notes: "Da die Tortuosität mir von keiner morphologischen Bedeutung, sondern eine Alterserscheinung zu sein scheint, habe ich sie nicht statistisch untersucht."



the two areas clearly and should be more useful here (Fig. 4). Should there be, in addition to an eroded carotid canal, a petrous

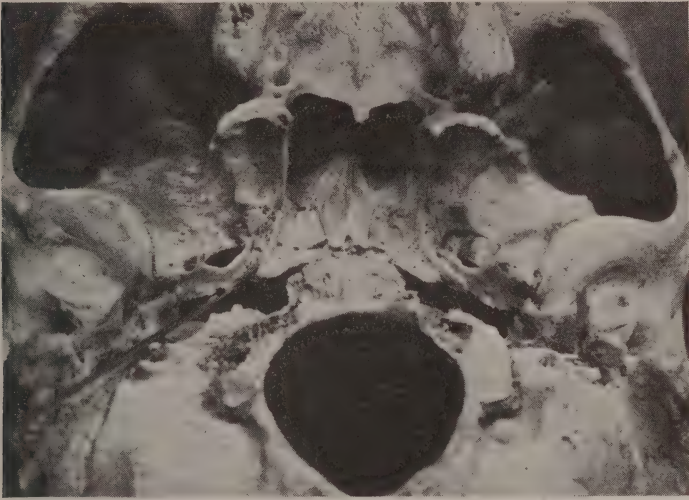


FIG. 4. Carotid canals converted into carotid groove by enlargement of the internal carotid artery. This view indicates the relation in the mentovertical roentgenogram.

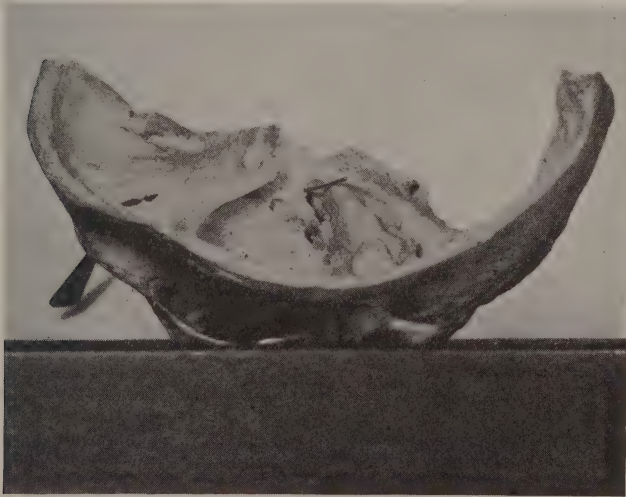


FIG. 5. Probe passing through dehiscence (posterior) in enlarged carotid impression.

apex infection, it should be remembered that this enlarged artery makes smaller an already tiny operative field.

Unfortunately the many turns of the internal carotid artery have not been named. This makes the description of abnormal turns difficult. The normal carotid does not lie above the floor of the sella turcica when the skull is viewed in roentgenograms, or in the anatomical laboratory. Under the condition of increase in length under discussion, this is not true. These extensions toward the vertex, when partially calcified, are seen by the roentgenologist. The otolaryngologist finds interest in the specimens in which the bend in the internal carotid artery, between the foramen lacerum and the



FIG. 6. Probe passing through dehiscence (anterior) in enlarged carotid impression.

side of the sella, has so enlarged medially that the bony wall of the sphenoid sinus has been eroded (Fig. 5). This erosion is in direct line from the pyriform orifice through the sphenoidal ostium. Anteriorly the same dehiscence may occur (Fig. 6). Here the loop of the artery, as it bends forward and upward from the sella, has exposed the sinus. The clinical significance of these exposed vessels is obvious. The difficulty of making the differential diagnosis between tumor and enlarged carotid artery will not be discussed.

The sphenoidal sinus is crossed anteriorly by the sphenopalatine artery. I am aware that Canuvt and Terracol (1925)\* minimize

\* Footnote, p. 206: . . . nous n'avons jamais été témoin d'hémorragies graves dues à la lésion de l'artère sphéno-palatine. La tradition, tout comme aux époques préhistoriques des dragons fabuleux ou de Minoture, transmet oralement des accidents mortels survenus surtout à l'étranger ou à l'âge de pierre rhinologique.

this vessel as a source of severe hemorrhage. However, I have frequently seen here an abnormal, large, displaced vessel. This, when examined in the light of our discussion, is seen to be a pathologic condition involving many vessels rather than an anatomical anomaly. The large vessel sometimes encountered on the floor of the nose may fall into the same category.



FIG. 7. Diagram of principal vessels of the head and neck displaced by arteriosclerosis and hypertension. The arrow indicates that portion of the artery causing erosion of the carotid canal.

### SUMMARY

1. Of the many anomalies of the vascular system of interest to the otolaryngologist, some, the true anomalies, are not readily forecast.

2. A large group, which we might call pseudo-anomalies, are here shown to be due to pathological changes in the vascular system. Their presence may be suspected in cases of peripheral arteriosclerosis, especially when associated with hypertension.

3. A diagram of these pseudo-anomalous vessels here discussed is presented as if found in one individual (Fig. 7).

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## DISCUSSION

SAMUEL IGLAUER, M.D., Cincinnati, Ohio: It is rather presumptuous for a mere laryngologist to discuss a purely anatomical dissertation; however, Doctor Batson requested that I should do it.

The essayist pointed out that Thessalius found a certain bone in the foot which later proved to be an anomaly. Anatomists, together with Doctor Batson, pointed out that Rembrandt's "Lesson in Anatomy" shows a tendon in the arm which should not be there. Perhaps Rembrandt has been unjustly criticized, since he may have been dealing with an anomaly.

The essayist did not refer to the venous system. However, we should consider the question of a jugular bulb protruding into the middle ear, the difference in size of the internal jugular veins, and also the difference in diameter of the two sigmoid sinuses. The thyroidea ima vein when present and distended, may interfere very seriously with tracheotomy.

The essayist has spoken of vascular anomalies, which might be termed acquired rather than congenital.

While we are all aware that a sclerotic artery is tortuous, as manifested in the temporal region, along with the gray hairs marking time, we have not sufficiently considered that these arteries are necessarily elongated and always increase in diameter.



As to the practical application in performing surgical operations, we should always expect the usual rather than the unusual, and we should not forget that we have an educated finger as well as an eye.

In considering the relation of an abnormally placed innominate artery to the trachea, I understood the essayist to say that we might find it in adults but probably very seldom in children. Tracheotomy should usually be a deliberate operation, but in those instances where we have to perform a penknife tracheotomy in an emergency, it is better to open the trachea high up, even at the risk of injuring the vocal cords.

The practical application of a dilated carotid in the carotid canal, of course, has great significance nowadays, wherein the petrous bone is so—shall I say—frequently invaded.

The other practical application is that in doing surgery in any part of the body you must figure on the common thing and not look for the anomaly, but as pointed out by the essayist, given a patient with signs of arteriosclerosis, such a patient may have anomalous distribution of the main arterial trunks as a result of the arteriosclerosis and therefore it shows again the intimate link between internal medicine and specialized medicine, which I think we too often forget.

NORMAN PATTERSON, F.R.C.S., London, England: I have been suddenly called upon to make a few remarks on this very interesting contribution. I have been extremely intrigued by these anatomical variations. As far as I can see, you have to chance the dividing of one of these rare vessels in a tracheotomy that has to be carried out in a hurry.

Then the external carotid, passing under the angle of the jaw here, might be in jeopardy in doing a tonsillectomy, or in operating for a malignant growth in that region.

The relationship of the abnormal internal carotid to the sphenoid is of interest. With regard to the sphenopalatine vessel, I know of a case—it happened in a friend—where there was a large hemorrhage in opening the anterior wall of the sphenoid. The patient nearly died.

With regard to penknife tracheotomy, in any urgent case I think it is always advisable, if possible, to do a laryngotomy rather than a tracheotomy, and then you are free from any danger of opening the large vessels.

There is one condition I should like to remark on and that is an abnormally large mastoid vein. Mr. Cheetle many years ago read an extremely interesting paper on that. Apparently the mastoid vein may be as large as the internal jugular, the venous part going through the cortex and passing down into the external jugular.

I have met a case in which apparently there was no internal jugular vein on the one side, simply a vein near the carotid. I wonder what would happen if one had to tie the opposite internal jugular for sinus infection.

JOHN F. BARNHILL, M.D., Indianapolis, Ind.: I wish to discuss some of the points which Doctor Batson has gone over. Of course, there is no discussion about facts, and I possibly can only add to some of the facts that he has already mentioned.

I wish first to speak of the anomalous division or bifurcation of the carotid arteries. It is usually stated in our textbooks that these carotid arteries bifurcate on a level with the superior rim of the thyroid cartilage. Now, that is a matter of

considerable practical importance to us at the present time, for, as you well know, the laryngologist is shifting inevitably into the neck in his work.

It has been our observation that the superior carotid artery bifurcates almost as often above the hyoid bone as it does in the region ordinarily given in our standard textbooks on anatomy, and it is a very common thing indeed for the external carotid artery to bifurcate under the angle of the jaw and even higher up.

In a recent observation of twenty-three cadavers, thirteen of these had arteries which bifurcated above the hyoid bone, and two of them had arteries which bifurcated under the angle of the jaw. This would be of tremendous importance to anyone who was attempting to ligate this vessel.

Speaking of the internal facial artery as it passes through the sphenopalatine canal, I wish to confirm what Doctor Batson has said, that this artery is sometimes of enormous size, and of all the arteries we find almost anywhere, whether in young persons or old, this artery sometimes is enormously curved and kinked, and gives the observer the feeling that he is looking at a worm that has tied itself into a knot just in front of the sphenopalatine ganglion, and this has an importance because one may need to go further back than the artery which nearly always lies in front of the ganglion, and it is nearly always larger than we think for it is nearly always curved and knotted in the way I have spoken of.

As a practical thing I wish to speak for a moment of the veins of the neck. Our distinguished guest asked, "What would happen if you ligated the internal jugular on one side when it was almost absent on the other?" Your patient, Doctor, might recover. There are a number of instances in which both jugulars, as you know, have been ligated, and the patient lived.

We have not given the attention to the emissary veins which they deserve. Our distinguished guest has just mentioned the enormous mastoid veins that are sometimes present. Another vein to which we should give more attention is the pterygoid. I fancy few of us have ever thought of the pterygoid plexus and pterygoid veins, but when we are ligating the jugular veins we are absolutely safe if we happen to have a splendid return of circulation to the pterygoid, and we will save the patient, whereas, if the opposite obtains, we may have ill luck.

FRANK ROBERT SPENCER, M.D., Boulder, Col.: I should like to put on record at this time a case in which the external carotid artery was underneath the right tonsil. It was very tortuous and very large. The patient died from severe infection and the postmortem showed the very large vessel.

It seems to me not improbable that many of the cases who die from tonsillar hemorrhage may have this anomaly. We were able to have a postmortem in this case to prove the point.

OSCAR V. BATSON, M.D., Philadelphia, Pa. (closing): The anatomist has always described dead arteries, and has assumed that they were live arteries; however, we have very recently had a report, I believe by Doctor Churchill, of Boston, in which he was able to measure a patient's aorta at operation and again able to measure this patient's aorta at autopsy, and there was considerable discrepancy in the diameter, because in life even the diameter of the artery is considerably distended and lengthened because of internal pressure. You can determine that for yourself by taking a strip of fresh artery from the butcher and using Alexander's syringe, put pressure in the vessel, and see what happens in the matter of length and diameter.

Just because an artery does not appear in your operative field in the cadaver does not mean that a slight increase in length will not push it to the operative field in the patient. We shall have to revise all our notions about diameters of vessels because we have had no adequate study of that matter since Keen did it a good many years ago.

I am appreciative of the discussion of veins, also. It is a subject on which I am working at the present time and have made some preliminary findings, and I should like to mention, Mr. Patterson, that the English literature on the posterior condyloid vein seems to minimize it. Perhaps it is because you do not have the large number of negroes that we have here. There may be some racial difference, but we see a great many large posterior condyloid veins which are not constant, but I have seen as many as six cadavers out of ten showing these large veins, and they are frequently half the size of the internal jugular.

Perhaps that is one of the mechanisms for survival in case of ligation of the jugulars. If the patient has condyloid veins, there is a by-pass; if he doesn't have them, there is no by-pass. Perhaps that may be determined in radiographic examination, because some radiologists make that report when they do an examination of a skull.

## EARLY AND LATE OPERATION FOR ACUTE SUPPURATION OF THE MIDDLE EAR AND TEMPORAL BONE

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The problem of when to operate in acute suppurations of the middle ear and temporal bone is a subject obviously too large for comprehensive discussion in a paper of this sort. Too many important factors demand consideration. The significance of laboratory studies, such as spinal fluid examination, blood counts, determination of hemoglobin, and study of cultures taken from the tympanic discharge at paracentesis and from the mastoid at operation, together with bedside study of symptoms, charts, etc., all deserve attention. These are only a few of the factors omitted that have some connection with the subject.

Today I propose to discuss the problem of selecting the optimum time for operation in temporal bone suppurations from an angle that I fear has not yet been sufficiently stressed in the literature, *i.e.*, that early operation is contrary to sound surgical principles pertaining to the successful treatment of acute inflammatory processes when the end in view is drainage.

The importance of the timing factor may be influenced by the character of the pathologic condition for which an operation is planned. For instance, a plastic surgeon aims at repair of a defect or deformity. Usually such an operation is not done in the presence of progressive disease. The timing of the operation is, therefore, of little importance in such instances. In progressive diseases the timing of the operation is usually of great importance. The ultimate object of the operation may be eradication of the disease by excision and removal of the lesion *in toto*, as in benign and malignant growths, inflammatory diseases amenable to such treatment, etc. For instance, appendectomy is sometimes performed successfully at any early stage because the infection is implanted in an organ of no functional value which can be removed *in toto* before the infection reaches the peritoneum where it may be capable of spreading to a fatal termination. The entire temporal bone and its foci of infection cannot be removed and placed in a specimen jar as can an appendix. In other instances the operator attempts to cure by drainage of the inflammatory process or by a combination of partial excision and subsequent drainage.



Perhaps my own ideas on the surgical treatment of acute inflammatory processes are largely influenced by my having had nearly seven years' training in general surgery prior to becoming an otolaryngologist. I emerged from this training with certain general rules for surgical guidance thoroughly ingrained in my system. Some of these maxims I regard as golden rules of surgery and their importance has grown in my estimation throughout the succeeding years in which I have found them sound and reliable.

Wise doctrines of general surgery can usually be carried over bodily into the special fields of surgery, and among the above-mentioned axioms is one having to do with the successful management of acute inflammatory processes. Inflammation is the reaction of living tissue to injury and whenever the injury sustained is due to bacterial invasion and the resulting inflammation is of the type that usually terminates in localized suppuration, it is a good surgical principle to refrain from attempting to drain by any traumatic attack with instruments of cold steel until the localized stage of the disease has been reached. It never becomes a good risk for surgical drainage unless such a stage is reached. If the infection is of the diffuse non-localizing type and so overwhelming that the patient's resistance cannot exert some successful effort to combat or check it, then it never becomes a good risk for traumatic surgical attack and any rough manipulation is contraindicated. I know of few exceptions to this rule. Perhaps future advances in surgical technic pertaining to the use of galvano-cautery, diathermy or some similar coagulating or sealing-in method may demand that this rule be changed, but I doubt if any change will ever be warranted when applied to the use of knife, scissors, gouge, chisel, curette, rongeur, etc. In other words, the keynote of success in the treatment of fulminant inflammatory processes is gentleness, and if trauma must be used it is better tolerated after the infection reaches its peak.

Once an infection becomes implanted in the layers of any tissue containing no effective anatomic or other barriers to an extensive spread, its progress can only be inhibited by some successful effort of resistance or walling off on the part of nature. This must be true regardless of whether the disease is of a suppurative or non-suppurative type.

The first stage of inflammation is one of engorgement—a vascular and cellular hyperactivity. Later a better organized and more definite line of body resistance appears and the ultimate type of lesion begins to assert itself. Still later comes a more or less successful effort at

repair. The first stage of inflammation is seldom amenable to surgical drainage.

The only rational argument for early surgery in an acute inflammatory process must be that the type under consideration is exceptional in that better localization is present early than late (some types of septic meningitis, for instance).

Examples of the difference in surgical treatment required by different pathological processes are numerous. Their relationship to the problems under discussion may seem questionable, but to my mind it is definite and vital because all have to do with timing of drainage in acute inflammations.

A cellulitis differs from a well localized abscess in its pathologic character, and in its requirements for successful surgical treatment. Tissue in a cellulitis is hard, beefy and infiltrated. It should never be incised in the early stages. If permanent moist dressings under rubber tissue and rest of the part are employed at first it may require no further surgical treatment and if incision later is necessary it may be more successful than if done too early. At first, tissue of an abscess is hard and infiltrated, as in a cellulitis. It should be handled in the same way until a localized collection of pus has formed. A peritonsillar inflammation is at first a hard, beefy, infiltrated or markedly edematous swelling. It should never be incised at this stage—never prior to localization of pus. Occasionally a peritonsillar inflammation never localizes and remains a hard infiltrated cellulitis and as such should never be incised. Many will fade quietly away, if given the opportunity to do so without being roughly manipulated. If a peritonsillar inflammation is incised by mistake, prior to abscess formation, a tracheotomy set should be placed by the patient's bedside and kept there until all danger is passed.

Most inflammatory orbital swellings will recede without localizing as orbital abscesses, if given the opportunity to do so. Early incision only opens into beefy, bloody tissue of the cellulitis type. Hot moist packs, rest and a little time will abort most of them without operation.

A carbuncle can sometimes be excised en masse and the resulting wound may heal by first intention. I have never seen it done successfully until definite lines of demarcation between pathologic and healthy tissue had been established.

An erysipelas differs from a folliculitis, boil, abscess or carbuncle as a spreading streptococcus edema of the pharynx does from a well localized peritonsillar or postpharyngeal abscess. No man of sound surgical judgment would attempt to treat the two types of pathologic

processes the same way. An erysipelas due to a virulent form of streptococcus may spread from any given point over the entire cutaneous surface of the body. It never becomes a good risk for surgical drainage or excision. The only effective treatment is rest, supportive constitutional measures and soothing applications. I have seen cases of erysipelas in which the entire lesion was no larger than a dime, but so typical in its clinical appearance that a tentative diagnosis could be made. In one case the initial chill did not occur until twenty-four hours after the patient was first seen and the diagnosis suspected. I cannot conceive of any surgeon with sound judgment attempting to incise or excise such a process hoping thus to prevent its subsequent spread. He should suspect that the sharply defined edge of inflammation visible to the naked eye does not necessarily mean that the actual extension of the infection through the lymphatic is limited to that area. Such an attempt might succeed, but I would find it difficult to applaud the judgment of the man who tried it.

I concede that infectious bone inflammations may differ in some degree from similar conditions in softer tissues, but I do not grant that they must necessarily require very different surgical treatment.

I recall some discouraging cases of long bone infection seen during the years I followed general surgery. Often we thought they had been caught in time and early and radical operations were performed which eventually proved unsuccessful. I realize that these failures of early surgery in long bone infections do not prove that we were working along the lines of bad technic. In some cases we met success. In most of these cases only a slight raise of the periosteum and a superficial lesion of the outer layer of compact bone was found. If we had waited too long, perhaps only diseased bone and discharging sinuses through a shell of periosteum would have been left. I have often wondered, however, whether or not more localization would have been present if we had waited a few days longer instead of attacking the part as soon as the diagnosis was established. Dakin drip and Dakin bath will cure some old cases which have never had the advantage of early radical surgery.

One more valid argument against early surgery is that it tends to oppose another sound surgical principle, *i.e.*, careful investigation before operation. It takes time to investigate a difficult problem. Hurried surgery tends to unsound conclusions based on snap judgment. Thus a central pneumonia can seldom be found with a stethoscope during the early stages and it is often accompanied by signs of meningeal irritation (the so-called toxic meningismus). If a sup-

purative otitis media is also present, the patient may be rushed to the hospital and taken to the operating room at once, without x-raying the chest to eliminate the pneumonia, on the theory that meningitis is imminent and immediate operation imperative regardless of what else might be found by a thorough physical examination. Ether might be decided upon as the anesthetic of choice, especially if the patient be a child of three or under. I have faced this difficult problem and have had to resist no little pressure from others who wished to proceed without delay. Only a firm refusal to be hurried has kept me out of several embarrassing situations of similar nature.

Early or delayed operation in acute mastoiditis has excited more discussion, perhaps, than operative timing in any other bone supuration. Only a few phases of this topic can be discussed here. In a previous paper of my own<sup>1</sup> I devoted some space to topics more referable to specific mastoid problems, such as: the significance of abnormal anatomy, previous injury and disease, routes of intracranial invasion, types of inflammation and their postoperative course after early and late operation, bacterial virulence and type of epidemic, age of the patient, an attempt at better standardization of what is generally meant by the terms "early" and "late," and other factors. The general trend of that paper was against early surgery, although no attempt was made to lay down hard and fast rules.

I advocate the same general principle of conservative delay in paracentesis and operations for lateral sinus phlebitis, labyrinthitis, petrositis and brain abscess drainage, and I even suggest that probably the present ideas on early meningeal drainage of pyogenic meningitis may eventually be considerably revised.

Early paracentesis in acute suppurative otitis media has not in my experience, shortened the course of the disease or averted dangerous complications. I do not incise a drum membrane until I am fairly certain that the middle ear content is purulent. Lowered hearing, mild earache and moderate temperature with a slightly bulging drumhead are not always sufficient evidence of the urgency of immediate incision.

Labyrinthine symptoms of vague character do not establish the diagnosis of suppurative labyrinthitis just because a suppurative otitis media is also present, but even when the diagnosis is positive a little patient waiting will often result more favorably than hasty surgery.

The neurological surgeons recommend waiting for localization in brain abscess cases, a procedure in line with sound surgical principles.

On the other hand, extremely early subdural drainage for meningitis is advocated. An early attempt to drain the initial localized



focus of meningitis near its portal of entry sounds feasible enough, but is complicated by the difficulty of accurately locating this focus at the proper time. In many cases the surgeon may feel confident at an early stage that some intracranial complication is beginning and still be unable to say for hours or even days whether it is a meningitis, lateral sinus thrombosis or brain abscess. To name the type of lesion accurately and its portal of entry on clinical findings before the patient reaches the operating room or autopsy table is often a difficult diagnostic feat. Eagleton,<sup>2</sup> speaking of spinal or cistern drainage says, "If we are to be successful in the treatment of meningitis, we must abandon many of our preconceptions, for in the past all surgical treatment has been based too much on the impossible principle of drainage of the entire arachnoid circulation." Whenever I contemplate a drainage operation on the meninges, the picture always comes to my mind of attempting to drain muddy water from a fuzzy fibrous door mat. I wonder if future efforts to advance in meningeal surgery would succeed better if directed towards sealing-in around the portal of entry rather than towards drainage.

Extremely early operation is seldom urgently indicated in suspected lateral sinus phlebitis. Some men seem to feel that each chill is accompanied by imminent danger of fatal metastases. My experience has been that such a chance can be taken on the early chills with a fair degree of safety.

In acute mastoiditis the specific purpose of the operation is an important factor. When this purpose is the prevention of intracranial complications, a wise decision is plainly of paramount importance, but this is not the only urgent indication for operation that may suddenly and unexpectedly appear. If no other likely focus can be determined, a mastoidectomy may be indicated to remove the primary focus in a systemic infection. For instance, a hemorrhagic nephritis may suddenly develop in a case of mastoiditis previously running a mild course and showing no indications of potential intracranial extension. Nevertheless the chances of any operation accomplishing its intended purpose must be carefully weighed against the chances of producing other and perhaps more serious complications if the operative time selected is inopportune. I contend that many apparently urgent indications for operation are relative and the real emergencies are exceptional. I differ completely with the advocates of early operation who recommend this procedure on the assumption that it is the surest preventive of intracranial complications in the fulminating type of case. That is precisely the type of case in

which some of us, having more faith in a conservative waiting policy, think that radical and early surgery is most likely to fail.

The success of early surgery in acute inflammations of the mastoid is frequently more apparent than real, due to an unrecognizable factor of chronicity which has been overshadowed by an acute exacerbation. The fundamental pathologic process, resulting from such a combination, might easily modify the rules of procedure usually obtaining at a similar stage of a primary or initial inflammation. In other words, many apparently early operations succeed because they are, as a matter of fact, not as early as they seem.

The factor of prime importance in selecting the optimum time for operation in acute mastoiditis may be an estimation of the probable state of health or normalcy of the mastoid when the last inflammation began. The operative time should be influenced considerably by previous injury, disease or abnormal anatomy in the mastoid. A perfectly healthy and normal mastoid should put up a better and more prolonged resistance to a new infection than a defective one, regardless of whether the defect is congenital and anatomical, or acquired and due to unrepaired injury or disease.

Previous attacks of mastoiditis may never undergo complete repair and organisms retaining much of their former virulence may remain potentially encapsulated therein for a considerable period of time. Such pathologic conditions may be lighted up into dangerous activity quickly and easily by reinfection. Early operation is more justifiable in such cases than when the mastoid had never before been diseased or had recovered its normalcy by complete repair of a previous injury.

If previous disease or injury of the mastoid can be established or suspected, an estimate of the probable degree of repair prior to the attack under observation should be attempted. It is hardly necessary to stress the importance of x-ray pictures in this connection.

The number and severity of previous attacks of mastoiditis and the interval of time between the last previous attack and the one under observation may furnish a yard stick for estimating the probable degree of repair from prior disease or injury and thus aid in the visualization of the present condition of the mastoid.

If a long and apparently healthy period of time has elapsed between the last previous attack and the one under observation, then complete repair may have occurred.

If the last previous attack has been recent, the degree of repair following it may be slight and the encapsulated organisms extremely virulent, in fact, the attack under observation may be a continuation of the former. This is the type of case in which early operation is

most justifiable or may even be urgently indicated. To my mind, what often happens is as follows: The patient complains of a slight earache; its importance is ignored and no tympanic suppuration occurs; the mastoid becomes infected simultaneously, but goes on to suppuration; weeks later, perhaps, pus and infection find their way back into the tympanic cavity and a suppurative otitis media results, either rupturing through the drum spontaneously or requiring paracentesis. The history of otitis media obtained dates only from the onset of earache produced by the reinfection and, therefore, the physician is misled and believes that he is dealing with an infection which can only be a few days old in either tympanum or mastoid, but in reality he is dealing with a mastoiditis of many weeks duration. A surprising degree of apparently early mastoid destruction has been so explained many times in my experience. In many instances the history of that previous non-suppurative otitis media was elicited later when no amount of questioning could bring it out during the excitement attending the preparation for a hastily advised operation. I operated in such a case on May 1, 1935. I first saw the patient April 21. The history of a severe earache beginning only forty-eight hours previously was given by the parents. Under gas-oxygen anesthesia a thorough paracentesis was done and much thick yellow pus released. The mastoid was then tender, the auriculomastoid crease was flattening and pitting could be elicited over the bone above and behind the ear. No evidence of a furuncle in the external canal could be made out. I delayed until fluctuation appeared May 1. At operation a subperiosteal abscess rested upon soft porous bone which extended from the root of the zygoma backward over the external canal and downward below the level of Henle's spine. After the operation I again delved into the history. The married sister of the patient was then present for the first time. She reminded the mother that when the patient had spent a day and a night with her about three or four weeks prior to the onset of the last earache, she had complained of deafness and had cried with earache much of that time.

From this and similar cases we may conclude that, in estimating the possibility of previous attacks of mastoiditis, a definite history of mastoid symptoms is not necessary because, if a suppurative otitis media has occurred, considerable coincidental mastoiditis must be assumed, and it can never be excluded if a history of non-suppurative otitis media is obtained.

Kopetzky<sup>3</sup> describes an acute hemorrhagic and two types of acute coalescent mastoiditis, and maintains that the hemorrhagic type remains so to the bloody end. If I correctly visualize what goes on

in a previously healthy mastoid during the early stages of any type of infection, it is always red and hemorrhagic at the beginning. Of course that is largely conjecture on my part, because I never operate on any type early if I can avoid it, but if this conception is true and all such cases are at first similar in appearance, how can a premature operation determine which type the case is destined to become?

Whenever I have operated upon hemorrhagic mastoiditis I have frequently been uncertain as to when I had finished. In surgery we depend chiefly on the senses of sight and touch to guide our course. In a hemorrhagic mastoiditis 50 percent of my armamentarium (visual sense) is absent. No definite visual dividing line between the pathologic and the healthy is evident. I depend more upon the soft feel of the bone than its appearance in some cases, and if no destruction of intercellular partitions is evident, even that help is gone. The borders of the cavity I have made are frequently as bloody as the tissues I have removed. One does not feel justified in removing the entire calvarium or even going to the outer suture lines of the temporal bone in such instances. Besides that, I cannot dismiss the idea that I am insulting angry tissue and, perhaps, sending septic emboli into the blood stream. Also the instinctive impression persists in my mind that by crushing acutely inflamed tissue perhaps a retrograde thrombophlebitis of intercellular veins will be produced which may lead to the very meningitis or intracranial complications I wish to avert or that, perhaps, infection is being carried into healthy or only slightly diseased tissue and the resistance of that tissue is being reduced by trauma. If the operation is delayed until the signs of acute inflammation have somewhat abated, then I think perhaps more success can be anticipated.

An accurate preoperative differential diagnosis between the hemorrhagic and coalescent types of mastoiditis is certainly desirable. How can it be made? I must confess that I have often failed. I have seen cases with a persistent hemorrhagic tympanic discharge, running a very stormy course (high temperature, headache, irritability, intense mastoid pain, tenderness and photophobia), in fact with many signs and symptoms causing me to fear that intracranial extension had already occurred, quiet down and recover without operation because I waited and decided the operation could be postponed another day, and yet another. Some of these cases, with later operation, were types of coalescent mastoiditis. On the other hand, I opened a mastoid last month with a constantly purulent tympanic discharge that had never run an alarming course, but quietly per-



forated at the tip of the mastoid into the neck, as a Bezold's abscess, early in the fourth week, and still the entire mastoid was hemorrhagic. I never dreamed that case would be hemorrhagic. Yellow pus came only from the neck. The tip of the mastoid was soft, the cortex over the tip could be opened with the scissors used to free the superficial sternomastoid fibers, but the content of the tip cells was hemorrhagic. The postoperative course was more stormy than the preoperative, but at no time did I feel concerned about an intracranial complication.

In the previously mentioned paper on acute mastoiditis I made the following statement: "I have never seen a case of hemorrhagic mastoiditis in the fifth or sixth week of infection except when good and sufficient reason existed to believe that an acute exacerbation due to a new infection had recently occurred." Since making that statement I have seen three such cases in the fourth week and one probably early in the fifth week (the history in this case was a bit vague). I must admit, if no visible tendency to coalescence was apparent at that late date, I can see no reason to assume that the appearance of the mastoid would change a great deal in a couple of weeks more. I am not yet convinced, however, that they all die unless operated on early as Kopetzky maintains.

Kopetzky<sup>4</sup> has implied that when previous attacks of mastoiditis have produced a sclerotic mastoid, early operation is indicated because pus is more likely to spread in various directions and thus more easily invade the intracranial tissue. He makes the same argument against prolonged waiting in the aged, *i.e.*, their mastoids are more sclerotic. However, if the sclerosis is properly placed, it may also inhibit progression towards the brain and tend to bring the infection to the surface as it will probably follow the course of least resistance. I suggest, however, in defense of his position, that a mastoid which has been previously diseased one or more times may resist early operative trauma better than a virgin mastoid usually does, and that even though surgery is done earlier than is absolutely necessary, not so much harm may result. This is merely another impression of my own, however, and it may or may not be accurate.

Ziegelman<sup>5</sup> has recently made the following statement: "In temporal bones, a periostitis, osteitis, osteomyelitis or suppurative process with or without abscess formation, may occur either independently or in combination. These pathologic processes may assume innumerable terminal forms, chief of which may be mentioned rarefaction, sclerosis, necrosis, caries and sequestration. The final result depends upon the cellular character of the temporal bone, the resist-

ance of the individual, the part and type involved, the method of invasion, the character of the infection present and the type of therapy used. We must go back to our surgical anatomy and the lesions that may occur in the temporal bone, and apply the principles of general surgery to the therapy of such pathology." I have attempted to apply those principles in this paper.

Waves of radicalism come and go periodically in all branches of surgery. Early operation goes hand in hand with radicalism in the surgical treatment of progressive diseases. I believe that a study of the history of medicine will disclose few instances in which radicalism has stood the test of time and survived. Usually the pendulum swings back to the final adoption of more conservative measures. In any branch of surgery one should fight against the idea that all diseases coming within the ken of the surgeon can be cured by early and radical operation. During my early and enthusiastic years I was apt to explain bad results as due to delay or insufficiently radical procedure. In non-surgical diseases the same inclination to blame conservative medication for failures may have to be conquered, but it does not take so long to discover the fallacy of a blind belief in the all powerful affect of drugs. In surgery it takes longer to struggle out of this fascinating haze, but sooner or later the importance of timing should begin to dawn on the surgeon and he should realize that faultless technic during the operation will not compensate for bad judgment in the selection of patients suitable for operation or in the timing of the surgical attack. In other words, the surgeon has more at his command than skilful fingers and all diseases designated as surgical are not to be solved by early and radical surgery.

In closing, my chief thesis is simply this—no fool-proof rule for selecting the optimum time for operation in acute suppurations of the middle ear and temporal bone is possible. No dogmatic rule can take into consideration all the urgent indications for operation that may appear in the picture at various periods of the disease. One may use good judgment and operate opportunely or use bad judgment and either operate too early or wait too long. In the final analysis nothing succeeds like success, and second guessing offers little consolation, but sound surgical principles should be studied meticulously, learned thoroughly and followed carefully when no distinct or irrefutable indications of when or what to do force themselves upon the scene. "When in doubt, wait!" is a safer policy to my mind than, "when in doubt, operate!" Sound precepts are valuable guides when the operator is otherwise in doubt, but the fate of any individual case of acute suppuration of the middle ear and

temporal bone depends primarily upon the sound judgment of the surgeon in charge, and this comes to the average man only through intensive study, excellent training and long experience.

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### DISCUSSION

HAROLD I. LILLIE, M.D., Rochester, Minn.: It is obvious that it would be difficult to prepare a comprehensive paper on this subject because so many factors not readily reducible to words, are taken into consideration when deciding upon the optimal time for surgical interference in infectious middle ear and mastoid disease.

The author has presented certain points that are debatable.

Ballance has said that when the pathology of any lesion is well understood, there should be no lack of agreement as to the course to pursue in the rational management of the disease.

Myringotomy is instituted to relieve the pain and afford drainage of the discharge caused by the middle ear disease. In secretory otitis media, it may be deferred; in suppurative disease it should be performed when the diagnosis is made. Myringotomy cannot be expected to prevent surgical mastoid disease in all cases. Myringotomy is preferable to spontaneous rupture because the incision is less liable to be followed by a permanent perforation of the drum.

The infectious processes that involve rigid wall cavities are quite different pathologically from the infectious processes that involve soft tissue structures. The structural differences in temporal bones are clinically and roentgenographically recognizable. The pathologic processes for the various types of structures are quite different. Operation in the coalescent type of mastoiditis, that occurs in well-pneumatized mastoid processes, may be more safely deferred than in the osteothrombotic type of disease that most frequently occurs in the diploic type of mastoid process. The clinical course is quite different. In the osteothrombotic type, if operation seems urgent, because of untoward symptoms and signs, nothing short of complete operation to non-bleeding bone will be effective. Incomplete operation will do more harm than good.

I hold a different opinion in regard to the urgency for operation in the subsequently reinfected cases. The fibrosis resulting from the previous infection may act as a good barrier to extension, although the subjective symptoms may be

greatly increased because the swelling of the tissue in the confines of the cellular structure is more intense.

If one should immediately operate on every case that presented certain signs said to be significant of mastoid disease, it is difficult to see how clinical or surgical judgment could be developed or relied upon, because the surgeon would never have observed the natural course of the disease.

No hard and fast rules can be laid down, as it is not a mathematical problem. Common sense, combined with individualized surgical judgment, will be the determining factors in choosing the optimal time for surgical interference.

JOHN F. FAIRBAIRN, M.D., Buffalo, N. Y.: Among 694 patients operated upon for acute mastoiditis on my service at the Children's Hospital, there occurred eight deaths, one from cavernous sinus thrombosis, five from meningitis and two from pneumonia. With the exception of scarlet fever patients, very few patients were operated upon within the first two weeks after onset of symptoms, and the majority were subjected to operation only after nature had manifested definite operative indications.

HILL HASTINGS, M.D., Los Angeles, Calif.: I think those who have practiced a number of years have found that in some years the virulence of an infection changes, just like measles, which will be epidemic and very severe for a number of years, and so, as we all know, will influenza, and that, I have found, after twenty-five years of experience, is true in our line of work. Some years we are persuaded that we must operate a little earlier than we would at ordinary times, due to the fact of the activity of the infecting organism in that particular year.

The other point I want to bring out is this. It has been my experience, and no doubt that of many of the rest of you, that some patients with an acute ear running three or four days manifest a very high temperature, and children will have at night what the mother thinks is a delirium, probably due only to the high temperature, and there is an insistence on the part of the internist or the surgeon for you to do a mastoid operation. They have ruled out all other possibilities, so there is nothing but the mastoid left.

In two cases in which I was about to operate—one patient I had in the hospital—it turned out, after a good pediatrician and internist examined the patient time and again, with negative findings, that there was a little rough spot in one lung, and I decided to wait. In the morning the pneumonic process was quite evident, though up to that time there had been no signs or symptoms of pneumonia.

We want to watch carefully for the deep-seated unrecognized pneumonia. I also feel if we operate on those cases, as has been done in the past, a pneumonia will develop and it will be laid to the ether. I think it was not the ether. I think it was an unrecognized pneumonia from the beginning.

VIVIAN W. WOOD, M.D., St. Louis, Mo. (closing): I do want to stress Doctor Hastings' point about pneumonia. A firm refusal to let anyone hurry me has kept me out of situations like that; very often we have to stand the pressure of a good internist and a good pediatrician, and they want to rush the patient to the hospital and operate without an x-ray, on the theory that the operation has to be done and that it is imminently necessary.



# THE NASAL ACCESSORY SINUSES AS A FOCUS OF INFECTION—EVALUATION OF VARIOUS DIAGNOSTIC METHODS

By W. P. WHERRY, M.D.

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I have elected to discuss diagnosis, the phase of this symposium assigned to me, from a broad viewpoint, perhaps a philosophic viewpoint, rather than to confuse the issue by presenting a mass of examination detail with which I know you are all familiar.

A completed diagnosis to my way of thinking includes, aside from a description of what is wrong, a concise summary of what you are going to do about it; in other words, your advice to the patient, and an analysis of the expected end-result.

To satisfy the definition of a diagnosis of chronic sinusitis, my experience has taught me that I would best be quite certain of three major phases, namely:

1. Identification of the pathology;
2. The measure of the infection load;
3. The patient.

I shall discuss each separately.

## IDENTIFICATION OF THE PATHOLOGY

To create a basis for argument, I offer the following conclusion: Strange as it may seem, even though modern surgery is based on pathologic premises, there are many surgeons professing special ability as otolaryngologists who are not clinical pathologists. Lack of this requisite is, legally, hardly acceptable when they qualify as specialists; and I can see no reason why this lack should be construed among ourselves in any other manner than as an inferiority of knowledge.

The identification of gross pathological changes in the paranasal sinus mucous membrane becomes the first requisite of a good diagnosis. In this connection, the title limits my discussion to chronic changes.

Accepting the dictum that chronic deviation from the normal in nasal and paranasal mucous membranes may, or may not, be pathologic, implies reasonable ability to evaluate changes other than those labeled as pathological.

According to Semenov, these variables should be noted as physiologic until such time as pathologic changes ensue, and, since the time quotient cannot always be the definitive factor, it should follow that recognition of frank, chronic alteration of tissue elements reaches

paramount differential importance. True, textbooks and the literature of yesterday attempted to emphasize this fact; such conclusions were, however, based on postmortem studies, which oftentimes had not the advantage of a sequential history. It is only since modern research on living tissue has been so thoroughly developed that a new histopathologic text on the diagnosis and treatment of chronic sinusitis in its various phases can be compiled upon a basis of scientific accuracy.

One has but to read the recent writings of Mosher and his co-workers—Schall, Knowlton, McGregor, Ross and others; of Fenton and Larsell; of Dean and his St. Louis group; of Sewall; and of Semenov, to realize this changing concept of the basic causative factors of rhinologic histopathology. For instance, the normal paranasal sinus membrane is about  $\frac{1}{10}$  mm. in thickness. The epithelial surface is ciliated, and in the subepithelial tissues are found few glands, few lymphocytes, a very few mesodermal cellular structures, and no sign of edema or leucocytic infiltration. The more recent work of Latta and Shaw tells us that there should be no goblet cells. However, in response to irritants or to physiological imbalance, there is an immediate reaction in the defensive mechanism, creating specific changes usually characterized by dominant factors. These include eosinophiles to combat allergens or foreign proteins; histiocytes, lymphocytes and polymorphonuclears against infection; water-logging of the tissues in the physiologic errors of toxemias, high or low pH; imbalance of the sympathetic and parasympathetic nervous systems.

As a part of my examining board experience, I have observed many records with tissue changes noted as hyperplastic. This statement seemed to express the pathologic diagnosis. May I emphasize the fact that inflammation is a perfectly normal defense reaction of a membrane to an irritant, whether bacterial or chemical, recent or of long standing; and only in the presence of the so-called sequential changes, such as beginning fibrosis or cystic degenerative areas, can the change be labeled as pathologic. The presence of a fixed transitory edema simply serves as a lead to probable associated causative factors which obviously in themselves direct the analysis elsewhere than the paranasal sinus region.

It must be admitted that readjustments in the accepted pathologic classification of chronic sinusitis are constantly being made necessary by the wealth of experimental results appearing in the literature. Not the least of the more recent assumptions is that some allergic sensitiveness is the probable basic reason for a mucous polyposis. Although such an assumption can alter the more or less classic diagnosis and approach now assigned to mucous polyps, the fact never-

theless remains that such a mucosal state should be labeled as a major allergic phenomenon only where the gross pathologic changes are of a minor nature. In other words, the diagnosis should evaluate the dominant factor at the time one's opinion is offered, since corrective measures, although embracing all factors, must start with the dominant.

An additional variable to be reckoned with in making one's diagnosis and prognosis is the inherent difference among individuals in resistance to infections, as well as the probable end-results of continued infections as to local pathology. To quote Semenov:

"On examining a large number of specimens, we find many individual variations in cell morphology. Some are robust, which will heal spontaneously, often being insulted for many years; others so undeveloped that from the day of birth they are predisposed to pathological processes. This leads one to believe that persistent sinusitis with retrogressive changes can sometimes be attributed to the rudimentary character of the mucous membrane itself. This concept that the sinus mucosa is a *locus minoris resistentiae* can be supported histologically. Once the soil is broken by vitamin, endocrin, allergic, vasomotor and other physiologic errors, the pathologic process is perpetuated and degeneration becomes inevitable."

Possibly one of the more likely errors of diagnosis in a full and final estimate of the sequence of paranasal sinusitis pathology concerns the presence of changes in adjacent regions. I refer primarily to the alveolar region. Much of the discredit that has come to paranasal sinus surgery can be accounted for by failure to find pathology in this region.

At one time we rechecked one hundred consecutive cases in which an external (Caldwell-Luc) operation upon the maxillary sinuses had been done. In twenty-two of these there was evident osteomyelitis of the alveolar region, one or both sides. In seven of the twenty-two cases the alveolar pathology was handled first with uniformly good end-results; while in the other fifteen the alveolar status was left for subsequent observation. Nine of these fifteen were reoperated upon. I was able to identify five of the fifteen as those which had drifted into other hands.

In recent years I have marvelled at the number of instances where seemingly frank nasal pathology has been corrected by cleaning up only the alveolar defects. Unfortunately few oral surgeons know how to handle this region surgically. From the oblique position of Rhese, we can estimate reasonably well the status of the teeth, well enough to secure additional roentgenographs if this area looks suspicious.

The danger of overlooking adjacent pathologic areas in an intranasal approach has prompted some otolaryngological surgeons to

routine advocacy of the external radical route. This rule may develop a better surgical percentage of cures in a large series of cases; but I feel that such a routine will surely lead to diagnostic carelessness, subjecting many individuals to unnecessary surgery, and perhaps increasing the distrust already mentioned.

I maintain that no examination directed towards a diagnosis is sound or can be considered thorough unless the nasal mucosa has been shrunk and anesthetized, and thus made ready for mechanical investigation of the area. I have found that this procedure allows a better estimate of nasal pathology, of possible bone changes, and of the apparent status of the ostia of any suspected sinus.

In this connection, transillumination prior to and after shrinking is oftentimes helpful in maxillary sinus studies. I am tempted to voice an opinion that transillumination, in maxillary sinus studies, even though discredited by some, is of value according to the degree of study exercised in its use. To me it is invaluable, especially where a time period for continued observation is possible.

Gentle suction irrigation with Locke's solution, where the suction as directed to the paranasal sinus is less than a quarter of a pound of air pressure, is very helpful in an estimate of drainage and the retained débris. When this is used in conjunction with transillumination, the information gained oftentimes supplements nicely that obtained by other means during the examination.

A clinical study is, of course, eventually incomplete without good roentgenograms. I used the qualifying adjective "good" advisedly. To permit the roentgenogram, however, to be the sole arbiter of an opinion, is not, I feel, good practice. Too many variables exist in the picture, such as a poor position, poor timing, poor development, plus the realization that in allergic membranes the water content will reflect a more definite shadow than will sometimes be noted in the presence of fixed pathology. In fact, I am willing to emphatically state that anything other than a perfect roentgenogram has no place in the diagnostic routine. I am also of the opinion that an interpretation of a roentgenogram in other hands than those of one thoroughly familiar with paranasal sinus pathology is very likely to be erroneous.

The use of lipiodol has become in recent years definitely valuable, both in visualizing the state of the membranes, and, in part, in estimating drainage facilities. One must, however, realize that lipiodol has a therapeutic potential. Seminov tells us that usually an edematous membrane indicates the presence of histamin in the tissues—an end-result of faulty cell cleavage; that lipiodol is the most potent of all the reagents experimentally used to dissolve histamin, there-



fore, lipiodol as a diagnostic agent sometimes requires repeated studies before the pathologic ratio can, with certainty, be made.

Antral punctures in times past were almost a routine practice as a part of a maxillary sinus diagnosis. In the past few years I have wondered why. The absence of staining or a discharge in the returned fluid does not always tell the state of the sinus mucosa. Antral puncture does not increase drainage facilities and can very positively damage an already insulted membrane. Repeated punctures surely will increase the pathological potential.

One has only to use the ordinary process of observation in a long series of the Caldwell-Luc approach to realize the damage that can follow. I suspect it is well to qualify this frank statement by saying that an antral puncture is sometimes warranted as a temporary therapeutic measure and occasionally becomes necessary to create confirmatory diagnostic evidence. Routinely used, however, I feel the procedure is unsurgical, believing that if it becomes necessary to puncture the naso-antral wall repeatedly, an opening of sufficient size to drain the area is indicated.

In recent years we have set up a system of recording conclusions numerically, thus: 1-1-1, or 2-4-3; indicating in order, at the time of examination; the pathology and its variables; the drainage state; and the patient himself. This is, of course, a mechanical system, probably only of use to those who arbitrarily established it. Its advantage lies in its compulsion upon the operator to sit down with himself and record a completed diagnosis upon which he is willing to stand, thus partly avoiding the danger of jumping at conclusions, or assuming facts that have not been fully determined.

It is indeed a human trait to accept each hypothesis presented in the literature as a fact, and to allow the most recent one noted to dominate one's reasoning processes, forgetting that after all the basic principles of pathologic changes are devious, and that two similar states are not always dependent upon the same factor. Therefore, any check that makes necessary the proof of one's conclusions becomes worth while.

#### THE MEASURE OF THE INFECTIOUS LOAD

The factor in view, suggested by the heading, is a factor without consideration of which good otolaryngology as a unit of general medicine, cannot be attained.

It matters not what term is used, be it focal infection, or just measuring the infection load, in the final analysis that patient with a low order of defense, or in whom, through long-continued or oft-

repeated insult, the defense is worn out, will react in distal regions that in themselves become an entity. Just whether this spread is caused by toxins or direct hits, is debatable, yet the fact remains that a correction of the initial process will in some instances relieve the distal manifestation.

Incidentally, a patient comes to his physician because he is in trouble and he should be considered as a problem to be solved, not just as someone to be worked upon.

Not infrequently it will be found that the paranasal sinus error is only one of several, that it may in part be eventually classified as a minor rather than as a major factor in the problem, and as such will possibly require a delayed observation until the vicious circle has been broken elsewhere.

Recently, experimental studies have disclosed the effect of changes in the pH and alterations in the heat potential upon the nasal and paranasal sinus mucosa. Likewise, the harmful effect of indiscretions in treatments and other items indulged in by patients, if undiscovered, can subsequently prove to be quite a major phase of the problem in hand.

Mention has been made elsewhere of the relationship of physiological errors and the paranasal sinus mucosa. The expectancy of the primary effect of such errors is quite different in a normal membrane and one already long insulted from other causes. Even as two and two make four, evaluating the problem in hand, if it is noted as allergy one, pathology three, obviously the pathologic factor becomes the major one and deserves correction first, and the allergic control should follow. The conclusion and advice in other words is determined by balancing in an arithmetical way all the major and minor factors.

The final word upon the "Focal Infection Hypothesis" has not been said but will some day be properly placed to clearly define what is meant by the term. At the present time, most of us have constructed our own answer from personal experiences plus the written experiences of others. I expect the final answer in so far as upper respiratory infections are concerned, will come when, associated with the conclusion, will be a statement of the thoroughness of the diagnosis and the accuracy of the correct approach—an audit of the operator, so to speak.

It has always seemed to me absurd, as well as academically unsound, to compile statistics wherein the controls and the variables were unknown. Needless to say, policies and practices are sometimes determined from such statistical studies. A case in point

is Mrs. "C," age forty-two, referred by an internist. The patient gave a history of frequent asthmatic attacks, always associated with, as she had noted, a bad cold. The exacerbations were frequent. She was a woman of considerable means and had been unable to maintain a proper surveillance over her affairs.

She was apparently physically sound, save for the asthma and the paranasal sinus pathology, which seemingly was confined to the ethmoid and sphenoid. A Mosher approach to the ethmoids and sphenoids was done, subsequently known to be incomplete. A respite from the symptoms was apparent for a few months only. A later examination noted the ethmoid pathology again apparent. A second operation, taking down as much of the sphenoid wall as possible, was performed with similar results, and at no time had the asthmatic-like attacks really been controlled.

A third approach surgically removed the entire mucous membrane revealing a perfect cast of the ethmoid and sphenoid. Sections showed extreme fibrous areas, cystic abscesses, numerous goblet cells and glands. A careful postoperative program was followed with stimulation of the operated area, using 2 percent iodine solution and an occasional silver nitrate application. A new membrane grew from which a biopsy taken eight years later showed ciliated epithelium and only an occasional goblet cell. The significant part has been the cessation of all the bronchial symptoms.

From the viewpoint of statistics and focal infection, this case was a liability after the first and second attempts and an asset only, following the external approach. A critic today would have charged the first eleven months' care in the error column.

Not infrequently untoward processes manifest a phase that prompts a most careful analysis and the diagnostic conclusion calls for a plan of action along lines developed from a series of collected cases rather than a single experience. I suspect that each of you, because of pressure exerted by correlated forces, has attempted the solution in a way contrary to your better judgment. A negative result proves only that someone probably made a mistake, yet added to the records of your own series or a collected group, it can be a liability where possibly the proper approach might have become an asset.

Good surgical principles define removal of a focus as a removal *in toto*, and does not countenance a partial or incomplete approach. The work of Mosher, Hilding, Fenton and others clearly points the way.

Analysis of the patient's environment—his epidemiology—is an essential phase of the gross study of his infection load. This factor

has been most apparent in observations both upon private patients and those in our university clinic. Time does not permit discussion, except to say that we give this factor serious consideration in the final conclusion.

The measure of an infection load can be summarized only after a complete survey of all the erroneously functioning physical phenomena. Consideration of each as a major or minor factor assists in determining the approach upon a pathological or physiological basis. In this connection, the principle of maintenance of good drainage becomes paramount, whether used as a measure of diagnosis, a method of treatment, or the means of furthering kindly repair.

### THE PATIENT

A diagnosis can be too highly departmentalized, even as some universities build a curriculum. It is so easy to "take for granted" phases that although basic, can be forgotten. One of these is the question of emotion. Someone has said:

Emotion is just as real a part of our lives as the intellect; the usefulness and happiness in life depends in a large measure upon our emotional balance.

An emotional imbalance then may mitigate against the effectiveness of a perfectly sound reasoning as directed toward the "locum tenens" of the area under discussion.

Graduated between the two extremes—the constitutional inadequate as proposed by Alvarez on the one extreme and the lethargic type on the other, are to be found emotional types, each suggesting tangents that must be assessed.

Alvarez consolidated a large group of the so-called psychoses and labeled them "The Constitutional Inadequates." This classification includes emotional types and psychotics, who flit from here to there with exaggerated syndromes, many of whom will also have frank pathological states present in the paranasal sinus. Regardless of what one may do, if a sympathetic personality of the operator is injected into the procedure, there will be a temporary respite, but surely this is only temporary. Many of this group will have a long operative list to their credit.

If in the final analysis, a surgical approach is deemed necessary, it is advisable to have in association someone with a dominant personality, who can, during the subsequent period, attempt the correction of the emotional state. However, if eventually the patient proves to be a constitutional inadequate, the end-result from his viewpoint is far from that desired. Surgery in this group should be of an emergency type only.



A neurologist handles such cases through suggestion, possibly in the manner of some speakers who talk a great deal but say nothing.

The lethargic type of patient presents a syndrome usually quite out of line with the severity of the problem. Not infrequently this type requires far more study; there is a greater likelihood of many major and minor phases and it is not unusual for differences of opinion to arise among consultants in the estimate of the infection load and how best to approach the solution.

An otolaryngologist, who for the time being assumes the rôle of a psychiatrist and clarifies his diagnosis by an analysis of the patient, may spare himself embarrassing moments.

In conclusion, it would seem pertinent to the discussion to voice just a word or two upon the inherent attitude of the diagnostician himself. In my travels, I have noticed three types of otolaryngologist.

There is the first, who visualizes his case only as a surgical problem, the second, who condemns radical surgery, preferring to temporize with an incomplete approach, and the third, who realizes that he has before him a problem and attempts to analyze it with an open mind. The diagnostic methods used are of little moment when a prejudiced opinion is presupposed, but become of great importance to him of an open mind, since it is likely that he will, as he becomes proficient with certain methods, secure the same information as his confrères do with different methods, equally good. In other words, the diagnostic methods used are secondary to a broad horizon of knowledge gained through the assimilation of research facts and experiences constantly made available to him who looks for them.

It has been said that a poor result condemns the diagnosis.

#### DISCUSSION

H. P. MOSHER, M.D., Boston, Mass.: I fully agree with Doctor Wherry that without a reasonable knowledge of clinical pathology in any given case, you cannot make a prognosis worthy of a specialist and chance plays a large part in your operative results.

I especially recommend to your attention and study the quotation from Seminov, and its application to the tissue changes commonly classed as hyperplastic.

I accept, as Doctor Wherry does, the statement that many mucous polyps are a mucous membrane reaction to some allergic state. This, to my mind, however, is of minor importance compared with the general clouding of sinus mucous membrane which often occurs in allergy. It should be a maxim, therefore, to rule out allergic states before a final diagnosis of sinus disease is made.

He has a good word for transillumination. In this matter I feel as he does. Suction, if not too strong and especially if locally applied, gives definite

information at times and has a certain therapeutic value. About lipiodol I would say that if you have a first-class roentgenogram you do not need to use it. The iodine in it probably has a slight therapeutic effect. I cannot, however, speak from practical experience on this point. Its hookup with histamine is new to me.

I do not mistrust a deftly performed antral puncture. Carried out repeatedly and in a clumsy and harsh manner I agree with Doctor Wherry as to its possible results. It is a temporary expedient only.

I do not find alveolar disease in 22 percent of my cases. If it is found, it is dealt with first.

Doctor Wherry's mathematical methods of recording tissue changes—drainage—and the patient's reaction to the amount of disease present in the accessory sinuses, I have never used. If it helps him or anyone else to get the proper perspective of the case in hand, it is to be recommended. He has borrowed the idea with due credit from Doctor Law.

The second division of the paper deals with the infection load. I take it that this means the amount of damage which the infection is doing locally, or what distant organs are involved as the result of the toxins elaborated in the infected sinuses. In other words, he brings up the discussion of the old question of focal infection.

The following quotation from Doctor Wherry I heartily agree to: "Not infrequently the paranasal error is only one of several and may eventually be classified as a minor rather than a major problem." In other words, in making a diagnosis of focal infection arising from the accessory sinuses, do not forget the rest of the body. Nothing has brought greater discredit to the specialty of otolaryngology than the numerous mistakes which we have made in this matter of accessory sinuses and focal infection. We have played this up for more than it is worth. Why do we not pick on a chronic running ear as a source of focal infection now and then?

The author cites two cases of operation as examples of poor diagnosis on his part. In making this frank statement I feel that he has overstated the case against himself. In connection with this statement, however, he says that the external operation allows more thorough work on the ethmoidal labyrinth than the internal. The question of the type of operation to be used in sinus disease is not the subject of Doctor Wherry's paper, he is supposed to be speaking only of diagnosis. Nevertheless, his offside remark is a natural slip. I am glad that he made it because in spite of my early labors in behalf of the internal nasal operation on the ethmoidal labyrinth I have no hesitancy in saying that the external operation is safer, and safely can be made more thorough. My feeling is that it will in great part supplant the internal operation.

The two pages which Doctor Wherry devotes to the subject "The Patient" deal with the great importance of the patient's emotional balance. He says, "An otolaryngologist, who for the time being assumes the rôle of psychiatrist and clarifies his diagnosis by an analysis of the patient may spare himself embarrassing moments." No matter what the pinch of poverty is the young practitioner should never forget this advice.

Again I quote, "If operation is deemed necessary it is advisable to have in association someone with a dominant personality who can correct the emotional state. However, if the patient grows to be a constitutional inadequate, in other words, a hopeless neurotic—the patient is never satisfied with the results. Only emergency surgery should be done on this type."

## THE RELATION OF ACCESSORY SINUS INFECTION TO GENERAL MEDICINE

By TREVOR OWEN, M.B.\*

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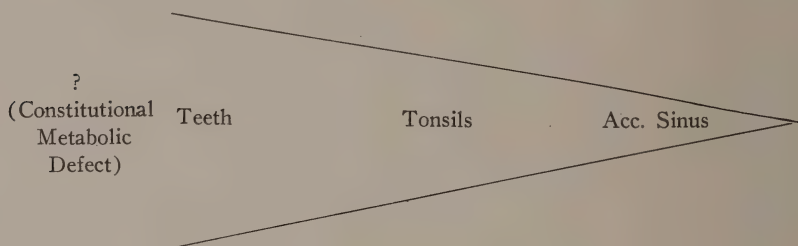
The large surfaces of the nasopharynx and mouth provide by far the most common entrance pathway of the body for organisms which share our environment. Commonly the human being is more pathogenic to them than they are to him. Whichever succumbs to the effect of that contact demonstrates a deficiency. If the human organism recovers completely from an acute infection, his state was one rather of unpreparedness than of defectiveness. However, if a chronic inflammatory state exists, in spite of an adequate blood supply and a good exit for waste products, then his organization is truly and basically defective. The multiplicity of, and the dissemination of, these chronic changes more and more denotes a serious defectiveness in his fundamental biochemical processes. These faults are due to hereditary, environmental and nutritional factors of which we have meagre knowledge as yet. The most unusual caution, therefore, must be exercised in scrutinizing narrowly *all* the factors in chronic illness.

In acute sinusitis the patient suffers from the same constitutional symptoms as in any acute infection. This is due, presumably, to a diffuse cloudy swelling of all tissue cells from their perfusion by blood containing a toxin on its way to be excreted or destroyed. But the absorption of bacteria, growing in the sinus culture ground in particular, and their transportation alive to distant tissues to grow there and produce inflammatory changes, are, in my opinion, most uncommon. There are many cases in which it seems clear that an acute upper respiratory illness is followed by what we term an infectious arthritis. In some of these, a sinusitis with insufficient drainage is found remaining after the rest of the nasopharyngitis has healed. In the majority no significant sinusitis is found and it is then true that these are milder and of shorter duration. The type of case where proper drainage of an antrum can be expected to produce recovery are those in which there is a clear history of an abrupt cessation of long continued perfectly normal health. In my experience the fewer the joints involved, without relationship to the severity, the more satisfactory are the results. While one cannot feel too sure that the sinus infection was

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\* By invitation.

responsible, in the first place, for the arthritis one feels hopeful that in some way it has been responsible for its continuance. This applies also to those cases of chronic ill health in which the prominent symptoms are easy fatigue (usually improved by rest), loss of weight, strength and appetite—symptoms often wrongly described as characteristic of tuberculosis somewhere, whereas they are simply the symptoms of a chronic infection of which tuberculosis is merely one. But the important problem is that of the old chronic sinusitis and its relationship to other local or sytemic disease. I believe that as a sinusitis becomes chronic it becomes more and more of local significance and the absorption from its surface of negligible importance. In the first place, speaking generally of foci of infection, one would put the accessory sinuses in the position, relatively to the other favorite areas, as shown in this diagram.



That is to say, where there really is a focal source of infection, the teeth come first, the tonsils a poor second, and the sinuses a most unsatisfactory third. Their treatment must rest on their own merit. I do not believe that chronic sinusitis has caused inflammatory lesions in the lung parenchyma or the heart muscle or valves, that it has anything to do with gastric or duodenal ulcer, cholecystitis, appendicitis, ulcerative colitis, nephritis, nephrosis, pyelitis or primary or secondary anemias. Of course, arthritis in its various forms is the big field for desperate remedies, and in rheumatoid and osteoarthritis when polyarticular, the removal of the so-called foci of infection is a most constantly discouraging experience. One feels that these foci of infection are the result of a constitutional defect rather than an additional cause. Their eradication does not improve or allay that fault, and sometimes indirectly makes the patient worse. There is no doubt, however, that a grossly infected sinus will add a burden of toxemia to a patient suffering from another chronic complaint but the question of treating that sinusitis should again be judged on its own merits. This is true in patients with degenerative diseases, such



as arteriosclerosis, thyroid dysfunction and diabetes. The arteriosclerotic stands infection badly but should be treated conservatively. The hypothyroid or myxedematous patient while not subject to infection, in my experience, is always made worse by local infections and a chronic purulent sinusitis will depress the production of what thyroid secretion they have and increase the severity of the disease. Diabetics are most sensitive to infection and their tolerance is much diminished, but diabetes is most certainly not caused by infection from the sinuses. On no account must treatment be carried out until the insulin dosage is entirely adequate.

In hyperthyroidism, on the other hand, foci of infection in my opinion should be left alone until the problem of the hyperthyroidism is solved. There are many mild cases of true hyperthyroidism in which the temptation is great to ascribe that condition to obvious infections in the teeth, tonsils or sinuses. To yield to the temptation of removing these foci of infection first in the hope of improving the hyperthyroidism is to invite almost certain failure and sometimes disaster.

Of the relationship of toxic and infectious lesions of the eye to sinus infection, I am not competent to judge but one's impression is that it may be very close. While not so hopeful in cases of choroidoretinitis, yet in retrobulbar neuritis and in toxic or infective neuritis of the 3rd, 4th or 6th nerves, a sinusitis may be the etiological point.

Patients who are subjects of allergy and who have vasomotor rhinitis and secondarily infected sinuses provide the large majority of chronic nasal invalids from repeated well-intentioned attempts to alleviate that condition. Their last state is far worse than the first. In asthmatics Warner and McGregor<sup>1</sup> have shown that radical antral operations relieved the asthma for two weeks after which, at varying intervals, all the patients relapsed. One can most seriously endorse their conclusions that attempts to relieve asthma by the radical treatment of a sinusitis are worthless. This is also true, on the whole, in cases of so-called chronic bronchitis. "So-called chronic bronchitis" because, in the first place, it would be unusual to substantiate that diagnosis pathologically and, in the second place, many of the patients alleged to have chronic bronchitis in reality have parenchymal lung lesions, or bronchiectasis. As the term chronic bronchitis is so loosely used to include all patients who have a chronic cough of any type, whether they have sputum and rhonchi and râles or not, there will be some whose cough reflex is initiated in the pharyngeal and antral mucosa from infection there. However, the quality of the cough distinguishes it from that of true clinical chronic bronchitis.

In the latter, the patients have a chronic cough with sputum, which is expelled from the trachea, and at some time or other have rhonchi and râles. They have no parenchymal lung lesion and no bronchiectasis. The large majority of these have no sinus infection, and in those that have a chronic sinusitis, no benefit can be expected from treatment of it. In bronchiectasis, the multiple dilatation of the bronchioles is subject to exacerbation of reinfection. We are most uncertain what are precipitating factors of this infection because while there is a constant stream of bacteria present in the inspired air, only at certain times does actual reinvasion take place. The presence of a chronic sinusitis or the occurrence of an acute sinusitis does not coincide with the lighting up of bronchiectatic inflammation and in the chronic bronchiectatic inflammatory state it is most unwise to hope for improvement by sinus treatment.

More than once in the course of this paper you may have felt that perhaps I was somewhat on the offensive. But the attack is not upon you but upon all of us. For while, on the one hand, operations are performed far too freely on the sinuses, as well as on the abdomen, in the face of continual and repeated failure, and the patient is made worse in mind and body—to say nothing of his estate—yet, in many instances, the physician has failed to tell you of all the factors in the case or has himself failed to appreciate them. Now one must mention that factor which most often trips us all up. That is the psychic factor. What a different turn is given to a case of chronic ill health with various somatic symptoms when a serious psychological problem is unearthed! So long as human nature persists in the belief that the mind is outside the body, so long will patients and their relatives continue their search throughout all the organs for the source of their chronic illness. Though they may have many subjective and some objective symptoms and signs, these will fall into their proper perspective only when all the facts of that individual's existence are exposed. It is up to us all, therefore, to be doubly sure of the ground on which we tread, first by insisting on knowing that all the possible factors in each particular case has been ascertained and, secondly, by drawing conclusions as to the apparent success of treatment only after a considerable lapse of time—again not losing sight of all the circumstances which may have influenced that success.

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## DISCUSSION

WILLIAM MITHOEFER, M.D., Cincinnati, Ohio: In the first paragraph of Doctor Owen's paper, he mentions two very important factors pertaining to the study of nasal accessory sinus disease—namely, the constitutional characteristics of the individual, and the fact that the nasopharynx is very often the gateway for the infection which secondarily affects the nasal sinuses.

We all probably agree with the assertion that the most difficult, as well as the most interesting, problem before the medical profession today is the study of a constitutional dyscrasia. Contact infection is never possible unless the constitution of the individual is ready for it. What change occurs in the cellular elements of the body to make the individual react unfavorably to certain irritants? Do the irritants come from without, are they confined locally within the nose, or do they originate within the body itself, as the result of defective metabolism? In other words, what is it that makes the soil fertile for bacterial invasion, or predisposes it to allergic reactions?

It is a known fact that a patient afflicted with a gouty arthritic diathesis is hypersensitive to certain foods, and if we believe with Gudzent that gouty manifestations, whether located about the joints or in mucous membranes, are allergic in nature, then some of the changes occurring in the mucous membrane of the upper respiratory tract will be better understood. Our chief aim must be to ascertain, if possible, the underlying cause of the allergy, and it is my firm conviction that the gouty arthritic state is one of them.

It has also been observed that a patient who has lived many years without showing any allergic manifestations becomes allergic following a severe infection, such as a nasopharyngitis or a pneumonia. If we remember that such is the case, we may be better able to explain some of the symptoms which manifest themselves during the period of convalescence; as, for instance, an infectious arthritis explainable on the basis of a bacterial allergy. If a patient becomes allergic following a severe nasopharyngeal infection, we may well expect a nasal sinus disease, which occurred as a complication of the nasopharyngitis, to run a protracted course. If such a patient be sent to a favorable climate without treatment, the sinus disease may disappear. This is probably caused by a change which takes place in the constitutional elements of the patient, as a result of the stimulating effects of the climate. There are many factors embracing the study of the constitution of the patient, and the abovementioned ones are but links in the chain. To be able to detect the offender is the problem.

I cannot agree with Doctor Owen when he says that a chronic sinusitis has nothing to do with gastric or duodenal ulcer, cholecystitis, etc. The constant swallowing of purulent material (acid in reaction) may readily be a contributing factor in various gastric disorders, including ulcer. The achlorhydria from the ingestion of a serous discharge (alkaline in reaction) also lays the foundation for a gastric, as well as intestinal, derangement. Zander has shown in an analysis of the stomach contents of nasal sinus patients that there was often an absence of hydrochloric acid, and that on this account flagellated parasites entered the stomach from the small intestine. Many of his patients complained of an indefinite pain and distress in the stomach. Hippocrates was probably correct when he said that stomach catarrhs have their origin in the head. In his work on "Airs, Waters, and Places" we find it stated, "Their

bellies are subject to frequent disorders owing to the phlegm running down from the head."

We have frequently seen patients in whom there occurred a mucous colitis following an acute flare-up of a chronic antrum disease. Whether this was a part of the general infection, was metastatic in origin, or was the result of an irritation of the vegetative nervous system is a question which cannot be satisfactorily answered. Appendicitis is often encountered in patients with nasal sinus disease. General surgeons will tell you that during a so-called "influenza epidemic" acute appendix inflammations are more frequent than at any other time of the year.

I agree with the essayist that hypothyroid patients, especially those of the benign type (of which there are many) are made worse by chronic purulent sinusitis. Such patients should never be operated on for nasal sinusitis without first ascertaining what is the tolerant dose of thyroid extract which they are able to take. The required daily dose of thyroid extract should also be given in the postoperative period. We should always remember to start thyroid treatment with small doses and watch for minor signs of intolerance, such as acceleration of the pulse rate, increase of blood-pressure, elevation of afternoon temperature which has been subnormal, and watery discharge from the nose. It may take four to six weeks with slowly increasing doses before the tolerant dose is reached. We have known patients who required six to eight grains a day before any sign of intolerance made its appearance.

I hold the opposite view of the essayist, and feel that hypothyroid individuals are very prone to infection. Infiltration, as we well know, is a characteristic sign of thyroid insufficiency, and is the result of improper elimination of products of decomposition. When mucous membranes become infiltrated, they appear pale and edematous, there is established a "*locus minoris resistentiae*"—a suitable soil for bacteria and allergens. The infiltration of mucous membranes may assume small proportions and not be easily recognizable. It is in such patients that we must look for other general signs of hypothyroidism before excluding the benign type of thyroid inadequacy from the picture. That thyroid secretion is one of our chief defenses against bacterial invasion, and that it is a great activator of metabolism cannot be denied.

I cannot agree with the essayist when he says "attempts to relieve asthma by the radical treatment of a sinusitis are worthless." If he bases his conclusions on the report of Warner and McGregor, he is taking into consideration the effects of antrum disease only, as a cause of asthma; for these authors, in their series of cases, did not operate upon the ethmoid, sphenoid or frontal sinus. It has been our experience that the majority of asthmatic patients who have a pronounced nasal sinus disease require a complete operation on all sinuses if good results are to be obtained. This means that an antrum, external ethmoidfrontal and sphenoid sinus operation is many times necessary. We must not forget that the mere removal of a focus of infection is not sufficient treatment for an asthmatic. It is but a part of a very complex problem. There still remains a disturbance in the harmony of the vegetative nervous mechanism caused by improper action of the reticuloendothelial system, as well as the thyroid and adrenal glands. In other words, the patient still possesses an asthmatic constitution which may be corrected if he receives exercises with a sufficient amount of sweating, is taught proper breathing exercises with pro-



longed expiration, is given a diet as nearly free from salt as is possible, and is treated in his postoperative period with autogenous vaccine.

Nasal accessory sinus surgery is, therefore, but a part of a very perplexing problem which requires a careful analysis in each individual patient. There is a sufficient number of patients with mild thickening of the antrum mucosa, as shown by the instillation of lipiodol, who also have vasomotor rhinitis and whose bronchial mucous membrane is probably in the same state of vasomotor turgescence, who should not, immediately at least, have their nasal sinuses operated upon.

Our experience has taught us that intranasal operations on patients with vasomotor rhinitis are very satisfactory, especially if the nasal sinuses are but mildly involved. We do not hesitate to correct a deviated septum, remove posterior hypertrophies, resect the overhanging edge of a hyperplastic inferior turbinate, or infract the turbinates in allergic patients. Free nasal respiration must be obtained if we expect a patient to make use of proper breathing exercises. A question which will always be a difficult one to answer is the following: Is the nasal sinus disease a part of the allergic condition, or is the nasal sinus the primary seat of disease, and is the infection responsible for the bacterial allergy making itself manifest in a hypersensitive patient? A cytological study of the secretions is often a help in these cases. Our procedure in dealing with the asthmatic patient is to do a complete nasal sinus operation when there is much pathological change present, and to attack all sinuses if necessary. When mild involvement is present in the sinuses we prefer to correct the nasal obstruction, surgically or by means of ionization, and do not desist in our efforts until the patient has free nasal respiration.

The essayist remarked "that operations are performed far too freely on the sinuses." He is correct in this assertion. A careful analysis of each individual case is imperative. Then there is the other side of the question. There are too many septum operations, middle turbinectomies, intranasal antrum operations done on patients who require more complete operations on the sinuses. We may, therefore, expect nasal accessory sinus surgery to remain in disrepute until rhinologists become more universally agreed on the proper method of surgical approach.

## CONSERVATIVE AND RADICAL SURGICAL METHODS OF TREATMENT

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The differentiation of the treatment of sinus pathology into conservative and radical methods has been long recognized. The distinction is easily over-emphasized and often produces a rather ritualistic mode of thinking, even to the abuse of classifying specialists as being in the category of either reactionaries or radicals. The wise practitioner should be neither one nor the other, but should carry out his treatment based on the underlying pathological condition, using his knowledge and experience to select that method which will best promote the cure of the patient's ills.

In considering the treatment of sinus disease as it manifests itself in a focal infection causing metastases in other parts of the body, we are led to wonder first why some of the infections produce this phenomenon and others do not. We have all seen patients going on for years with a profuse purulent discharge and, beyond some local discomfort, giving no manifestation of any bad effects on the general health. In fact this type of disease seems to be the one least likely to cause any serious metastatic condition. On the other hand, some rather obscure type with scanty discharge and thickened, boggy membrane in an antrum or ethmoid may be a certain source of a low-grade degeneration in the vascular system and kidneys; or perpetuate a chronic inflammatory process in the delicate tissues of an eye. We have looked in vain, I think, for the cause of this discrepancy in the manifold variations of bacteriology. This would seem to be a logical field for further research, for surely there must be something specific in the different properties of germs which, with the same apparent morphology, produce such different results. The anatomy of the nasal sinuses with all its variations and the individual resistance offers some explanation of the phenomena. Also the virulence of the original infective agent with the care and treatment which that received, must be considered an important factor.

To emphasize the importance of the latter, I think you will all agree that an original head infection manifesting itself with a very acute coryza and profuse discharge will be more likely to clear up entirely than the low-grade streptococcal infections which produce

severe headache and prostration with very little coryza. This type is usually called grippe by the general doctor, and is treated by saturating the patient with coal-tar drugs. I have seen such cases sent to the seashore to recuperate with an antrum full of pus, and the prostration and prolonged convalescence explained as the aftermath of influenza, when in reality they have a purulent or degenerative ethmoiditis, slowly passing into a chronic stage. If the ears were infected and continued to discharge, something would be done about it; but the sinus infection is allowed to continue to be a source of danger to the victims the remainder of their life.

In examining all cases for a possible focal infection, it is important to elicit everything in their history bearing on the attacks of grippe or influenza which they have had, and especially to inquire about the convalescent period from such attacks. Where antrum disease is suspected, it is well to get accurate data on tooth infections in the upper jaw, even though the teeth have been long since removed. The time period from the original infection to the secondary manifestations is about as variable in head infections as it is in syphilis. Many years may elapse during which an apparent immunity was sustained, and then from some accidental illness or devitalizing influence, the immunity breaks and secondary symptoms occur.

It is very important therefore to treat acute conditions till they are thoroughly well and this will usually be accomplished by conservative methods. The present day vogue in home treatment of the common cold is quite an improvement on older methods. The almost universal use of some of the ephedrine preparations does a great deal not only in adding to the comfort of the patient, but also tends to maintain drainage and render the condition much less liable to become chronic. A revival of interest in the old treatment by mild doses of an opiate is quite general. A combination of codeine and papaverine,  $\frac{1}{4}$  gr. each, known as the Wisconsin cold treatment, is very beneficial and is probably a little more effective than the old Dover's powder. Local treatment, beyond the inhalations of hot steam with a little spirits of camphor added to the water, is not usually advisable in the early stage.

The specialist will most often be consulted for head infections when they are passing into a subacute stage. Treatment of these so-called "hanging-on" colds is very important. A local or general involvement of the sinuses will usually be found with purulent discharge. The membranes will be found swollen and boggy with some loss of tone in the vessels of the submucosa. Conservative local treatment consists in shrinking the membrane, washing out secretion

by some form of suction irrigation, using warm saline or mild alkaline solution, or the Mithoefer combination of three chlorides. There is an objection to too frequent use of aqueous solutions on mucous membranes, but, while I think they often are used too frequently by the patient, I am sure the removal of thick, tenacious, purulent discharge from the nasal mucosa and sinus openings is an imperative demand in rational treatment. The most bland non-irritating fluid is the one best adapted for this purpose. This should be followed by a local application of some mild astringent. There are various agencies used for this purpose and one should be rather careful of his selection, for since the researches of Proetz and others have shown certain drugs to be inhibitory of ciliary action, it gives us some notion at least of things to avoid. Fortunately ephedrine hydrochloride seems to be one of the harmless drugs and has no detrimental effect on ciliary action according to Lierle and Moore. The desired end of such treatment is to reduce the swollen mucous membrane about the openings of the sinuses and so promote natural drainage. This medication should reach the membrane above the middle turbinate and the sphenoethmoidal recess. The argyrol pack is used by many in routine treatment, but it is very difficult to place in a position to do any good in most of the cases and its benefit is largely from reducing the swelling in the mucosa over the turbinates. I have always preferred the postural method for the administration of such medication. With the head far extended, gravity will carry any solution to the proper area. The colloidal silver known as collosol argentum, I have found the most satisfactory astringent for this purpose, though a warm solution of freshly made argyrol does very well with the disadvantage that it is more apt to irritate for a few seconds and also is not devoid of the danger of producing argyria when used too liberally. Medication may even be applied to the inside of sinuses by the displacement method of Proetz, but the important area to be reached is the membrane about the openings of the sinuses. The only operative interference in treating sinusitis at this stage would be the occasional puncture of an antrum. Many of them can be washed out directly through the natural opening, and this should always be tried first. The direct washing of a sphenoid may occasionally be necessary but is not very satisfactory. Where a real blockage occurs in this sinus and one washes it out through the natural opening, the subsequent reaction to the cocaine and the traumatism seems to make the natural drainage less effective. This also obtains with the frontal sinuses and I do not believe that many ever attempt the treatment of an acute or



subacute frontal sinusitis by direct irrigation. When an enlarged middle turbinate is blocking the nasofrontal duct, the anterior end may have to be removed. I would delay this until the virulence of the infection has lessened and would not advise doing it if a culture showed the presence of Type III Pneumococcus.

Assuming now that we have tried all our remedies and the case does not recover, if the patient's circumstances permit, we may advise a holiday in another climate with general tonic treatment. These conditions are a problem; we know if they are allowed to go on, they will keep up a chronic focal infection with all its potentialities for subsequent trouble and every known remedy should be tried to prevent this if possible. Autogenous vaccines may be given a trial and good results have been claimed for the use of antigens prepared from the patient's infection. They may be used locally and given hypodermically. Diathermy as a method of treatment is still very experimental; even its advocates maintain that it must not be used where an undrained pus cavity exists. This is often very difficult to determine and when it does exist, establishing drainage is probably all that would be necessary anyway. Theoretically, diathermy would seem to be a rational mode of treatment, but should be used by one familiar with the method. My own limited experience with its use would not enable me to pass judgment. Recently good results were reported by its use in several cases of maxillary sinusitis by Larsell and Fenton. The infrared heat lamp used in the patient's home seems helpful in some cases and has at least one virtue, *viz.*, that it does no harm.

We have thus reviewed what may be called prophylactic treatment of focal infections and the same methods would apply as conservative methods in the treatment of chronic conditions where the primary focus has already produced metastatic phenomena. Most of these patients come to us for a diagnosis and if our finding is positive, we have to advise and carry out the treatment. Let us assume that we find an apparently localized condition such as involvement of an antrum with other sinuses clear. In this case our decision will be between intranasal drainage or a radical Caldwell-Luc operation. If the history suggests a pathological process of long duration, the most radical procedure will be advisable. This will be imperative if the infection started from an infected tooth. As so often happens, one is in doubt about the amount of degeneration of the mucous membrane. I advise an exploratory operation, that is, making a small opening through the canine fossa, then by direct inspection or with a pharyngoscope, determine accurately the procedure. In

some cases one can estimate that drainage will be all that is necessary, so one can make an intranasal opening and close up the outside opening without touching the lining membrane. This method facilitates the making of a good clean intranasal opening as well, since one is able to see through the small canine fossa opening and in the event of striking a large blood vessel, one has an opportunity of stopping the bleeding. I have very rarely made this exploratory opening that I did not find the pathology worse than was suspected and have gone ahead and cleaned out the whole of the antrum lining. The same procedure should be carried out on the other side if there is any suspicion of pathology there. The intranasal operation is more likely indicated where there is profuse discharge than where a low-grade process of absorption is occurring from thickened membrane. The prognosis in maxillary focal infections is excellent as it is possible in this sinus to thoroughly eradicate the disease. Regeneration of the membrane will occur if the cavity remains clean, but if infection occurs either superimposed or as a recrudescence of the old infection, the healing process and the final result will not be satisfactory. The cavity in such cases fills up with granulations and may remain as a focal infection necessitating a secondary operation.

When we come to consider cases with a general involvement of the sinuses, our problem is more serious, but even some of these may be amenable to successful surgical treatment. If the ethmoids are apparently part of the focus, an intranasal ethmoidectomy should be done as thoroughly as possible. A good x-ray will demonstrate the anatomy and give one an idea of how thoroughly this can be done through the nose. In nearly all of them, even with orbital cells, a pretty thorough exenteration can be carried out of the main mass of cells and at least drainage provided for the cells in the hinterland. Some advocate the external route entirely to eliminate the focus. This is a more pleasant operation, I admit, but renders your patient liable to subsequent trouble from blocked frontal drainage. If the frontal is badly diseased, of course the external route is preferable and the frontal can be dealt with by the Jansen-Lynch or Killian methods, depending on the size and anatomy of the sinuses. Strange to say, the frontals alone very rarely act as a focus of infection and even when combined with pathology in the other sinuses, are not probably much of a factor in the absorption phenomena. The operation on the ethmoids must always be radical as it is not possible to drain individual cells. A complete exenteration offers the only means of eliminating a focus in this region.

The sphenoid is one of the worst sinuses to deal with in that it will most often produce evil effects both locally and generally, if chronically infected. The diagnosis and treatment will present great difficulties. X-rays are a great help in diagnosis and in obscure cases one should pass a small canula through the natural opening. Then two or three drams of sterile solution inserted into the cavity can be withdrawn into the syringe for a culture and cell count. Repeated examination of the patient may be necessary to make a sphenoid diagnosis. I prefer to see them early in the morning before they have cleaned out their nose and examine the posterior region with a pharyngoscope. At this time a small quantity of pus may be seen issuing from the opening. Treatment of a sphenoid should be both conservative and radical. The opening in the anterior wall should be made as large as possible at the first operation and the patency of this opening should be carefully maintained in after-treatment. Unless the lining membrane has undergone complete polypoid degeneration, it seems better to leave it. If the membrane is completely removed, the cavity is prone to keep filling up with granulation tissue and it is very difficult to ever get it to heal. This seems much truer of the sphenoid than of the antrum. If the operation for drainage with repeated lavage does not clear up a sphenoid focus, some more radical procedure may have to be tried. The sphenoids with a deep pocket in the bottom, present such perverse anatomy for proper drainage in the ordinary postural habits of human beings that many of them will continue to discharge and play their iniquitous rôle of focal infection in spite of treatment. To overcome this bad drainage, it may be necessary to remove the floor of the sphenoid. This can be done with suitable biting forceps and is easier accomplished if the anterior wall has been lowered as far as possible in the previous operation. Where both sphenoids are involved and the openings are hard to keep open, removal of the front part of the partition between the two will eliminate any further trouble of inadequate drainage.

The postoperative care of sinus patients is important. Removal of excessive granulations with biting forceps is preferable to the use of strong astringents which are liable to injure remaining mucous membrane. I have never found any solutions of any benefit in postoperative treatment of an antrum or sphenoid. In the latter the membrane seems extremely susceptible to any irritant. Carbolic acid  $\frac{1}{4}$  to  $\frac{1}{2}$  percent in oil as suggested by the late Doctor Sluder is about the best kind of application. This can also be injected into an antrum and seems to facilitate healing.

Bacteriophage locally is advocated by some. I have used it on several patients, three of whom had a pure culture of staphylococcus aureus in the antrum. These seemed like ideal cases on which to try it and the result was negative. The same disappointment followed its use in other sinuses. I did not combine the local use with subcutaneous injection, so perhaps I did not give it an altogether fair trial. As with vaccines, its advocates always insist that to get results there must be a hundred percent adequate drainage. That may be so, but when one has arrived at such perfection of technic as to fulfill the latter condition in the nasal sinuses, he may safely leave the case to the natural agencies of recovery.

In conclusion I scarcely need to remind you that with all local efforts every attention should be paid to the general treatment of the patient. We know that patients get well of tuberculosis where no local treatment is used and this occasionally happens with head infections. The administration of vitamins with an increase of calcium in the diet is helpful and the patient should always be placed in the best possible hygienic conditions. The treatment of associated pathological conditions such as allergy or chronic infections in the teeth and tonsils should always be considered. As Doctor Dean taught us many years ago, the first treatment of sinusitis in children is the removal of tonsils and adenoids, and one can almost say the same of adults, though this alone in the latter will not produce as high a percentage of good results as with children, but will render the susceptibility of the sinuses to reinfection after surgery very much less.

With all that can be done by all known means to remove focal infections in the paranasal sinuses, there will always remain some residue of infection, and though many brilliant results may be obtained, one who works in this field well knows that an over optimistic prognosis is not warranted in cases of paranasal sinusitis of long duration.

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#### DISCUSSION ON SYMPOSIUM: "THE NASAL ACCESSORY SINUSES AS A FOCUS OF INFECTION"

EDWARD C. SEWALL, M.D., San Francisco, Calif.: I think the important thing that has been brought out this morning is the question of the relationship of chronic disease in the sinuses to disease elsewhere in the body.



I would say only that it seems to me reasonable that we should defer judgment on this very important matter until we have learned to remove this focus of infection.

I cannot see how the internist or the specialist can draw any inferences as to the relationship between chronic sinusitis and other diseases until the chronic sinusitis is cured. We speak of our operations as if they did what we intended them to do. I don't think they do excepting rarely. I think they are very poor, and Doctor Faulkner has expressed it very fairly, and if we are to operate, we should try to improve our technic until, fortunately, some other technical means may take care of the operating technic.

SAMUEL R. SKILLERN, JR., M.D., Philadelphia, Pa.: I think Doctor Owen is wrong when he says that the sinuses are a very poor third to the teeth. I feel, personally, that you can get just as much infection or toxemia from an intrasinus infection as you do from the root of a tooth.

W. LIKELY SIMPSON, M.D., Memphis, Tenn.: Just to discuss Doctor Faulkner's paper, it seems to me an important point in the simple antrum technic to make a large opening, with clean-cut edges, and not leave a lot of shreds hanging around, if you expect to do any good. This can probably best be done with a circular cutting punch. Traumatism to either the middle or lower turbinates often defeats the purpose of the procedure.

The intranasal ethmoid and sphenoid exenteration can usually be done satisfactorily without the removal of the middle turbinate. The cutting of the attachment of the anterior end of the middle turbinate as suggested by Halle and beginning the exenteration with the removal of the agger cells, as suggested by Mosher and Halle, gives a good approach to this region. Of course, it is a well-known fact that many ethmoids are often overlooked and also that some cells cannot be exenterated due to anatomical barriers.

The Luc-Caldwell operation has given me excellent results in most cases. The smaller the opening in the anterior wall, the better, if all teeth are normal, but by means of mirrors even though the opening is small, a thorough operation can be done, but the operative time is necessarily prolonged. If the ethmoid and sphenoid are to be exenterated at the time of the radical antrum operation, the procedure can be facilitated if done by the transantral route. The ethmo-maxillary group is often overlooked except by this route. At times some of the anterior ethmoids and some of the outlying ethmoid cells cannot be reached through the antrum. The sphenoid exenteration is especially satisfactorily carried out by this method. If there are no teeth, the entire anterior wall should be removed.

In those sphenoids with a tendency to close with mucous membrane, scar tissue, etc., the removal of the septum of the sphenoid and also the posterior part of the nasal septum and much of the floor of the cavity usually gives a good result.

I should like to emphasize that drainage only should be sought in acute and fulminating sinus infections. In ethmoid and frontal infection, with complications such as orbital abscesses, periostitis, or osteomyelitis, external drainage should be obtained without any attempt to do a thorough, complete operation. If an external opening with good drainage without closure is made, the prognosis is much better than if exenteration of the frontal or ethmoid is attempted. If a thorough, complete operation is indicated, this should be done when all

acute infection has subsided. Usually in one or two months this procedure is safe. Many a patient has lost his life by too much surgery during the time of an acute infection.

Intranasal surgery in the presence of sinus complications, as periostitis, or orbital abscess, is not as good as external drainage, and this is especially true in little children.

If the usual intranasal sinus surgery associated with the radical antrum has not been satisfactory, or if a very extensive, marked degree of sinus disease exists, or if there are anatomical barriers, such as very marked extension of the frontal and ethmoid sinuses, the conservative operative procedure is an external operation upon these cavities. The attempt to exenterate marked extensions of ethmoid cells intranasally is anything but conservative.

Exenteration of the ethmoid, sphenoid, and frontal through an external wound through which all parts of these cavities can be thoroughly inspected with the naked eye, or by means of mirrors whereby all pathological tissue can be removed with very little trauma to the bony wall of these cavities, is much more conservative than most intranasal procedures. The operation can be satisfactorily done under general anesthesia, but to me local anesthesia makes the entire procedure very much easier and safer. Scopolamin and morphin analgesia makes the approach to the procedure much more facile.

W. P. WHERRY, M.D., Omaha, Neb. (closing): I still believe that a good, thorough diagnosis is the key point and that no statistical studies and no conclusion should be allowed to answer a summary unless the approach has been a thorough one (and I am certain that all of us agree that only in the more recent years has our surgical procedure even approached thoroughness). I say when a focus is suspicioned as such, it should be labeled as a focus only when it has been removed *in toto*, not partially, and no conclusion should be set up until then.

TREVOR OWEN, M.B., Toronto, Ont. (closing): I retract nothing. And I feel that we are all in the same boat, and we are all in difficulties, and for various reasons.

I see these patients long after the nose and throat men and the surgeons have started, and I don't see how you could have told what to do, because, as I gather, the question of diagnosis is still a little hazy, and I agree with Doctor Skillern that when you have an osteomyelitis, and the sinus is not the only thing affected, then you are in the same position as when you have an abscessed tooth.

Now, we are all in agreement as to difficulty in the estimation as to what patients are going to be benefited, and in a statistical study you cannot find that out, but it is only by the careful investigation of each particular patient, but it must extend over many, many years, and I think Doctor Mithoefer has not put that quite fairly, because in questions of sinus infection in gastric and duodenal ulcer, if patients are followed over a number of years, with all other factors put in their proper perspective, he will find that the results are not coincident with the improvement in the sinus infections; and the majority, after all, of patients with duodenal ulcer, never have any sinusitis at all; and mucous colitis, on the other hand, is not a disease but a disorder, in which the patients are subject to that same kind of disorder which is apparent in the sinuses, and quite likely they will occur together.

Now, each patient is an individual problem and it is only by a long history that we can come to any conclusions. If a patient has a sinus which is

discharging pure pus, then I am interested in that sinus, but if he has a sinus which has some thickened mucous membrane, and has a few shreds of pus in the mucus, then I am not sure that I am interested in that. Isn't that an academic sinusitis? From an internist's point of view, I am not so sure that I am interested in that problem.

Of course, the whole thing is a matter of very careful consideration in every case.

E. ROSS FAULKNER, M.D., New York, N. Y. (closing): I can't say that I altogether approve of the ultraconservative attitude being assumed today not only by the profession but through the entire lay body in regard to sinus operations. We all know it is a very difficult surgical field and we all know it is almost impossible to eradicate focal infections where all the sinuses are involved; on the other hand, I think the bad repute of sinus surgery comes very largely from incomplete, badly done operations.

I should be very glad, if Doctor Owen comes to New York, to show a goodly number of patients which have been benefited, and many secondary operations done in places where you would think they had proper, thorough attention, especially in otitis, which Doctor Owen passes over, where the result is nothing short of startling and brilliant when you alleviate the sinus condition. The results in asthma are not satisfactory, of course, and yet some are very, very satisfactory taken as a whole.

A man has to be very careful in the selection of sinus cases with asthma, if he is going to operate, and there are a great many other things to consider. Opening an antrum isn't going to cure asthmatic cases where there are a lot of other sinus involvements. It is just stirring up infection and probably will do the patients more harm than good, but if you eradicate as thoroughly as possible, you will get very brilliant results in sinus surgery.

# DIFFERENTIAL DIAGNOSIS OF ENLARGEMENT OF THE LYMPH GLANDS OF THE NECK\*

By R. F. FARQUHARSON, M.B.

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Enlargement of the lymph glands of the neck may be classified in several different ways. For purposes of this discussion, the following classification is suggested:

## *Local Enlargement:*

1. Lymphadenitis secondary to infection, as: in tonsillitis, pharyngitis, herpes, aphthous stomatitis, furuncles of head, face and neck, infected abrasions of face, scalp and neck.
2. Tuberculous adenitis (rarely generalized).
3. Early Hodgkin's disease.
4. Lymphosarcoma (rarely generalized).
5. Secondary carcinoma (primary in face, head, neck, nose, sinuses, larynx and stomach).

## *Generalized Enlargement:*

1. German measles.
2. Syphilis.
3. Infectious mononucleosis.
4. Lymphoid leucemia—acute, chronic.
5. Advanced Hodgkin's disease.

## LOCAL ENLARGEMENT

*Lymphadenitis Secondary to Infection.*—Enlargements of cervical lymph nodes in response to local infection usually present little difficulty in diagnosis, especially when the primary lesion is acute. Everyone is familiar with the tender swelling of glands that occurs in patients suffering from furuncles, herpes, aphthous stomatitis, and acute tonsillitis and quinsy. Usually the swollen glands are discrete, seldom very large, almost always tender, often associated with periadenitis and slight inflammatory edema, and rarely of importance. They disappear when the acute infection subsides. Occasionally in scarlet fever and other acute streptococcic infections a more severe adenitis arises, the glands are larger, exquisitely tender and fre-

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\* By invitation.



quently fused in an inflammatory mass that may suppurate and require incision. The patient is ill, with high fever, general weakness and sometimes acute hemorrhagic nephritis. The acute character of the lesion makes the diagnosis obvious. The primary lesion may be insignificant, especially if in the scalp, but a careful examination of the area drained by the enlarged tender gland will reveal the source of the infection.

In the presence of subacute or chronic inflammation and in patients suffering from recurrent acute infection, especially of the tonsils, the regional lymph glands are often slightly enlarged, fairly firm, discrete, definitely outlined, either not tender or only slightly tender. Such glands give little trouble in diagnosis, but their presence is evidence of the local disease and an indication for tonsillectomy.

*Tuberculosis.*—Tuberculous adenitis is perhaps the commonest cause of gross chronic enlargement of the cervical lymph glands. Frequently one of the easiest diagnoses to make, it may at times be distinguished with difficulty from a low-grade septic infection, on the one hand, and from Hodgkin's disease or lymphosarcoma, on the other. Children and young adults are most commonly affected and especially in districts where the milk is not pasteurized. Characteristically, it begins in the glands at the angle of the jaw on one or both sides, the tonsils being the portal of entry. Increase in size is slow and for some time the patient may be quite unaware of the disease. It commonly extends to the submaxillary and superior deep cervical glands and later to the inferior deep cervical glands. It may be localized for long periods, remaining inactive and inconspicuous. With increasing activity, larger swellings appear, usually the greatest mass being at the angle of the jaw with enlarged glands of gradually decreasing size and varying consistency forming an irregular chain down the neck. The glands at this stage are tender and the patient suffers from malaise, fatigue and low fever. There is periadenitis with a certain amount of inflammatory edema which renders the outline of the gland indistinct and gives rise to matting together of adjacent glands and groups to form irregular masses of varying size. The inflammatory process may go on to softening and suppuration with involvement of the skin and ultimate breaking down and sinus formation. More frequently healing occurs and the swelling may subside to become imperceptible, or fibrosis and calcification may occur leaving definitely enlarged glands, firm to hard, sharply outlined, discrete, not tender, that give rise to no symptoms.

Occasionally in a patient whose tuberculous adenitis seemed to have been healed for years, the inflammation may suddenly become

acute and even suppurative, resembling subacute septic adenitis. Then history of former disease and absence of primary infection in the area drained are helpful diagnostic features.

Although tuberculous adenitis is most common in children and young adults, it may occur at all ages and sometimes a hard tuberculous gland in old people is mistaken for secondary malignancy. Although it arises usually high in the neck near the angle of the jaw, it may begin and remain localized in the supraclavicular lymph glands, especially those near the head of the clavicle, when the differentiation from Hodgkin's disease may for a time be difficult. At times also it involves chiefly the salivary glands and related adenoid tissue. Then it may be associated with uveal disease, giving rise to the syndrome of uveoparotid tuberculosis of which facial paralysis is a common symptom.

Tuberculous adenitis is easily diagnosed when the disease is active with enlarged tender glands, especially when these are high in the neck. There is usually no doubt of the condition when it goes on to suppuration or when it is recurrent. Difficulty arises when, as rarely, it is extremely widely spread or when there is a large, firm non-tender mass, particularly if low in the neck. Then it is to be differentiated from Hodgkin's disease and lymphosarcoma.

*Hodgkin's Disease.*—Hodgkin's disease, also referred to as lymphadenoma, lymphogranuloma, or malignant granuloma, is a chronic disease, most typical in adolescence and early adult life, that first manifests itself in enlargement of a local group of glands and later, after weeks, months or years, extends to involve glands in other areas. Most commonly the supraclavicular glands on one side are first involved, but the disease may begin in glands in the post-cervicular triangle, in the axilla, infraclavicular region, mediastinum, abdomen, or groin. At first the glands are discrete, freely movable, not tender, slightly firm and elastic. It is at this stage that differentiation from tuberculosis may be difficult. Later, they become firmer, may even be hard, and usually adhere to one another to form large masses of semidiscrete glands that are never fixed to skin or deeper structures and rarely, if ever, tender. Frequently quite a large mass of this type is to be felt in the supraclavicular area without material enlargement elsewhere; sometimes, on the other hand, groups of elastic, discrete glands with no single large mass anywhere may be felt in several areas by the time the patient seeks medical advice. From the first the patient usually suffers from lack of energy, fatigue, mild malaise, some loss of weight and low fever. Not infrequently distressing pruritus is an early symptom. Occasionally the tempera-

ture is moderately high and sometimes there are periodic bouts of fever lasting seven to fourteen days associated with aggravation of the general symptoms which abate in the interval. Early in the disease there is little or no anemia, although the patient is often pale. Later, a secondary anemia of moderate degree develops. At first the white blood count is normal; there is usually a slight relative lymphocytosis, often an increase in endothelial leucocytes, and not infrequently an eosinophilia. In the later stages, the white blood count rises, commonly to 15,000-20,000—in one instance in a patient at the Toronto General Hospital, it was 40,000—with a high percentage of polymorphonuclears.

When glands in many areas are involved, the diagnosis is usually easy. Lymphoid leukemia is ruled out by the absence of a high white blood count and the rarer aleucemic leukemia by the absence of a marked lymphocytosis. Sometimes widely spread tuberculous adenitis is confused with Hodgkin's disease and, indeed, many authorities believe that the two diseases frequently exist together. It is in the early stage when the enlarged glands are localized and discrete that greatest difficulty occurs. In Hodgkin's disease, however, the spleen is usually palpable from the first, whereas this is quite an uncommon finding in tuberculosis. Tuberculous glands are commonly tender and this finding at once rules out Hodgkin's disease. Tuberculous adenitis usually begins high in the neck and Hodgkin's disease almost always low in the neck. Sometimes, however, a group of non-tender tuberculous glands in the supraclavicular region is almost indistinguishable on physical examination from Hodgkin's disease. It is true that tuberculosis tends to affect glands near the head of the clavicle, whereas in Hodgkin's granuloma the masses usually appear well out in the supraclavicular area. One may resort to biopsy and in most instances the pathologist will be able to differentiate the two conditions. Not infrequently, however, there will still be room for a difference of opinion and the diagnosis will then be determined by the subsequent course of the disease. In patients suffering from Hodgkin's disease, glands in other areas will become involved, the enlarged glands will disappear quickly under radiation therapy to return, unfortunately, after a period of several months; and within a few years the disease will terminate fatally. In tuberculosis, on the other hand, the glands may enlarge further, become tender and even suppurate; usually the disease will not spread to other areas although the axillary glands may become involved; almost always the inflammatory process will subside, although it may recur even after many years, and very rarely will it be fatal. Occasionally,

however, especially in patients suffering from a generalized or miliary tuberculosis, there may be widely spread tuberculous adenitis with splenomegaly. It is in such patients that the two diseases are believed to exist together. In these relatively rare instances the granulomatous reaction in the glands to the tuberculous disease is of a type very similar to that of the Hodgkin's granuloma.

The clinical manifestations of Hodgkin's disease vary considerably as does its pathological picture. The more typical cases are found in adolescents and young adults. In older patients it tends to run a more rapid course, often associated with larger local swellings and sometimes with infiltration of neighboring tissues. Should a gland be removed, the microscopic appearance of such tissue may resemble that of lymphosarcoma. In some cases, according to Ewing, there is actually a transformation of Hodgkin's granuloma into a sarcomatous condition which he calls Hodgkin's sarcoma.

Because of the difficulty, at times, found in distinguishing between Hodgkin's disease, Hodgkin's sarcoma, lymphosarcoma, and similar diseases of lymph glands, many writers have grouped them all together under the term, lymphoblastoma, making no attempt at differentiation. The frequent occurrence of borderline cases affords some justification for doing so. There is, however, a very typical picture of Hodgkin's disease with fairly distinctive clinical and pathological characteristics. It is true that this shades off till it may be indistinguishable from tuberculous adenitis on the one side, and from lymphosarcoma on the other. Yet the differentiation is of definite value.

*Lymphosarcoma.*—Typically, lymphosarcoma is a local condition arising from a group of lymph nodes or from adenoid tissue of mucous membranes. Rarely is it generalized. Unlike Hodgkin's disease it commonly begins in the tonsil or adenoid tissue of the pharynx. There is progressive enlargement of the primary tumor with ulceration and extension to the regional cervical lymph nodes. The spleen is not affected. General malaise and fever do not appear in the early stages and there is no pruritus. The glands subside quickly under radiation therapy but, unfortunately, the recession is but temporary and the disease recurs and progresses, usually going on to a fatal termination in from one to five years.

*Secondary Carcinoma.*—Cervical glands may be secondarily involved when there is carcinoma of the mouth, tongue, pharynx, larynx, nose and nasal sinuses, ear, face, scalp, pituitary body and stomach. Usually there are but a few palpable glands and these are hard. With further growth they become fixed and ulceration of the



skin may occur. Occasionally quite a large mass of malignant glands is found.

In most instances the site of the primary growth is known and the nature of the glandular enlargement is then obvious. Occasionally a large, hard "sentinel gland" above the head of the clavicle, usually on the left side, is a helpful sign of carcinoma of the stomach which formerly may not have been suspected. Sometimes a transitional cell carcinoma (lymphoepithelioma) arises inconspicuously high in the nasopharynx, the enlargement of regional lymph glands being the first evidence of the disease. The primary tumor may be found only after most careful examination; sometimes only by careful palpation in the pharynx. In a few instances secondary carcinoma has been found in cervical glands when it has been impossible to find any primary disease even after most careful investigation. The finding of hard, non-tender cervical glands for which there is no apparent cause is, of course, an indication for excision of a gland and histological examination of the specimen.

#### GENERALIZED ENLARGEMENT OF LYMPH GLANDS

Enlargement of lymph nodes in many areas, as well as in the neck, is characteristic of certain generalized infections, notably: German measles, syphilis, and infectious mononucleosis, as well as of lymphatic leukemia and the later stages of Hodgkin's disease and some cases of lymphosarcoma.

*German Measles.*—One will seldom have difficulty in recognizing the adenitis associated with German measles. Beginning usually a day or two before the appearance of the rash, sometimes as long as a week before, there is a slight but definite and very widely spread enlargement of all lymph glands. Those in the neck along the posterior border of the sternomastoid and in the mastoid and suboccipital groups are perhaps most constantly affected and most easily palpable. The glands are small, firm, discrete and usually painless. Occasionally fairly marked enlargement may occur locally. Often the patient is unaware of the glandular swelling until it is drawn to his attention by the physician; but sometimes the glands are tender and they may give rise to stiffness of the neck before the appearance of the rash explains the nature of the trouble. The glands usually subside as rapidly as they appear, leaving no trace of the disease.

*Syphilis.*—In the secondary or generalized stage of syphilis there is slight, widespread enlargement of lymph glands which are small, firm, freely movable and not tender. They appear coincidently with the characteristic skin eruption and luetic pharyngitis and tonsillitis.

Rarely conspicuous, they often remain for some months after the disappearance of other secondary manifestations, then gradually disappear. Their importance is frequently exaggerated and often in thin people relatively normal lymph glands may be considered as suggestive of luetic disease. The finding of slightly enlarged lymph glands in the occipital region, provided one can rule out German measles and local lesions of the scalp is, however, suggestive of early luetic infection and demands further enquiry into recent history and serological investigation.

Lymph gland enlargement in late syphilis is very unusual. Sometimes gumma of the sternomastoid or other tissue may be mistaken for luetic adenitis, but on careful examination one will soon recognize the error.

*Infectious Mononucleosis.*—Infectious mononucleosis or glandular fever is an infectious disease of unknown etiology that affects children and young adults and, strangely, appears to be particularly frequent in medical students, nurses and young physicians. Usually it begins with malaise, fever, headache, sore throat, and early enlargement of cervical and other glands which are tender. Occasionally the onset is more gradual, tenderness of the enlarged glands being an early complaint. At its height the temperature reaches to between 100 degrees and 104 degrees. There may be marked swelling of the tonsils and adenoid tissue of the pharynx, sometimes with ulceration. The enlarged glands are almost always tender and the enlargement is usually greatest at the angle of the jaw. Swelling of submental glands is frequently seen and enlarged glands are usually present in each axilla and often in the inguinal and femoral regions. The spleen is commonly palpable and sometimes tender.

A striking feature of the disease is the increased white count which may reach to 15,000-20,000, or higher, in the second week of the disease, with a lymphocytosis usually of over 70 percent. There is, however, no anemia, no hemorrhagic tendency, no reduction in blood platelets. In the third week of the disease the patient begins to improve; temperature, pulse and white blood count fall toward normal; the glandular swellings subside and the patient gradually and completely recovers.

The presence of sore throat, fever, generalized lymph gland enlargement, high white blood count, and lymphocytosis naturally lead one to think of acute leukemia.

*Acute Leukemia.*—Acute leukemia, whether lymphoid, myeloid or monocytic, is a disease that progresses rapidly to a fatal termination within a few months. It is characterized by a gradual onset with

slight weakness, palor, increasing fever, generalized enlargement of lymph glands and spleen, ulcerative stomatitis and pharyngitis, hemorrhagic tendency, and increasing anemia. The white count is commonly high, but may be normal or even decreased; the great majority of the white cells are immature. The platelets are reduced in number. The gradual onset stands out in contrast to the more abrupt onset of infectious mononucleosis. Angina, an early symptom in glandular fever, is later in acute leukemia. In both, because of the tonsillitis and pharyngeal lesions, the glands at the angle of the jaw are larger than those in other areas and usually tender. In infectious mononucleosis there is no associated anemia, no purpura or hemorrhagic tendency and no reduction in platelets. Should one be in any doubt, a short period of observation will quickly clear the diagnosis; for the patients with leukemia will slowly become worse, whereas those with infectious mononucleosis will improve rapidly in the third and fourth weeks and completely recover.

*Chronic Lymphoid Leukemia.*—There is no other disease that gives rise to such widely spread multiple enlargement of superficial lymph glands as does chronic lymphoid leukemia. In all areas in the neck—submaxillary, submental, high and low in anterior and posterior cervical triangles, in the occipital and supraclavicular regions—one finds numerous firm, discrete glands, not tender, that vary in size from about 2 centimeters in diameter downwards. Wherever in the body one looks for lymph nodes, they will be found enlarged. The spleen is always palpable and sometimes quite large. The tonsils and lymphoid tissue of the pharynx are commonly involved.

Such universal enlargement of superficial lymph glands may be quite marked before there is anemia or any definite malaise or weakness. Early in the disease, however, the white blood cells are increased in number, usually to more than 50,000—commonly 100,000 to 200,000, and sometimes much higher. The type cell is a small lymphocyte.

Just as in Hodgkin's disease and lymphosarcoma, there are variations from the typical picture. The white blood count may be normal or but slightly increased, the percentage of lymphocytes being high. Such aleukemic leukemia is, however, quite rare.

Occasionally there is leucemic infiltration of surrounding tissues in a sarcomatous fashion. One may find infiltration of liver, pancreas, pleura or superior mediastinum. In some such cases tracheal obstruction may give rise to severe dyspnea with stridor. Cases in which leukemic infiltration occurs often run a subacute course to a fatal termination within a year. Usually the white blood count varies

between 50,000 and 100,000. When seen early, however, the white blood cells may be decreased—an aleukemic phase—but usually the count rises to high figures in the terminal stages of the disease.

As stated earlier, Hodgkin's disease is never so generalized as is chronic lymphoid leukemia. The blood picture serves further to differentiate the two conditions. Only rarely in grossly atypical cases will one have difficulty in distinguishing one from the other.

### DISCUSSION

The diagnosis of enlargement of cervical lymph glands is usually made without difficulty on history and clinical and hematological examination alone. Of the swellings affecting only cervical lymph nodes, difficulty is sometimes found in differentiating certain cases of tuberculous adenitis from lymphosarcoma and early Hodgkin's disease. A biopsy is often helpful in these circumstances but, unfortunately, the histological picture is not always decisive and the diagnosis may then be determined only by the subsequent course of the disease. In patients with generalized lymph gland involvement, one must be careful not to confuse infectious mononucleosis, which is never serious, with acute leukemia, which is always fatal. The differentiation is clearly made by clinical examination and study of the blood picture.

In conclusion, one would stress the importance of history and clinical examination with a consideration of the blood picture. The information obtained by histological examination of the excised gland is often useful, but sometimes indeterminate and occasionally misleading. It should be considered together with all the clinical data in the final summing up of difficult cases.

### DISCUSSION

C. STEWART NASH, M.D., Rochester, N. Y.: Doctor Farquharson has presented a clean-cut, concise differential diagnosis of enlargement of the lymph glands of the neck, and we are indebted to him for renovating this subject matter. Every otolaryngologist has in reserve some scheme of differential diagnosis of enlarged cervical glands which he brings forth as the occasion demands, but it is particularly fitting that this subject matter be presented from the broader point of view of general medicine.

This paper is not controversial; we accept his statements as facts. There are, however, two comments I wish to make. In the first place, he has not taken into his differential diagnosis agranulocytic angina, which sometimes produces associated cervical adenitis, and which in at least one case presented a problem of differentiation between an infected lung mononucleosis and an agranulocytic angina. Secondly, in his paper he makes the following statement, which I shall quote. He says: "Occasionally a large, hard, sentinel gland above the head of



the clavicle, usually on the left side, is a helpful sign of carcinoma of the stomach which formerly may not have been suspected." To me this sign is both interesting and unique. If sufficiently dependable, it gives the otolaryngologist an opportunity to make a very clever diagnosis on a malady somewhat astray from his own particular field.

LYMAN G. RICHARDS, M.D., Boston, Mass.: I should like to make one further addition in connection with certain congenital anomalies in the form of branchiogenetic cysts. These not infrequently cause difficulties in diagnosis and from time to time can simulate true cervical gland enlargement; and, finally, I should like to speak to the point of differential diagnosis between true cervical gland infection and signs and symptoms of these in conjunction with lateral pharyngeal abscesses in the neck.

We see these quite frequently located behind the posterial pillar of the tonsil and situated lateral to the constrictors of the pharynx, occupying somewhat that region known as the pharyngomaxillary fossa. Abscesses in this region, particularly in younger boys from eight to ten years of age, often present a diagnostic problem. The classical appearance is that of a swelling along the lateral pharyngeal wall, but frequently in conjunction with that there is an external swelling in the neck. It is somewhat difficult at times to estimate the significance of this external swelling. If it is regarded as part and parcel of the pharyngomaxillary infection, there is naturally a temptation to approach this surgically by the external route.

However, this external swelling, in my experience, is not infrequently due to a secondary glandular enlargement entirely apart from the actual abscess and located in the cervical glands in this region; therefore, it has been found very important to differentiate this condition, and to watch carefully the appearance of the lateral pharyngeal wall.

In case this infection begins to show prominence and bulging in this region, as it frequently does, the internal approach and drainage and incision from within the mouth will almost invariably evacuate this infection and not require external incision.

If such external incision is made in the acute swelling on the outside, when it is due to an acute cervical adenitis, disastrous results may follow. If, however, it is treated and drained from the inside, almost invariably the acute glandular swelling, the swelling on the outside, will subside of itself and not require incision.

This differential point, I think, is a very important one.

FREDERICK T. HILL, M.D., Waterville, Me.: In this beautiful, comprehensive, differential diagnosis I think we should keep in mind in addition the possibility of neoplasm in the nasal pharynx and the importance of routine examination for laryngeal pathology, and the possibility of a chronic lingual tonsillitis, especially in cases who have been tonsillectomized.

THOMAS E. CARMODY, M.D., Denver, Col.: Some years ago I reported two cases, one of them bilateral, of tubercular infection of the carotid gland. At that time I could find only eleven cases reported and one of them was a terminal case of tuberculosis and the other was a case quite far advanced.

I would like to ask which of these cases of Doctor Farquharson's were primary tuberculosis and which were secondary, and why they cleared up so quickly.

Another point we must consider is the infections from the naso-pharynx. We may have some other infection besides the one we are trying to diagnosticate,

because even with a differential diagnosis we may have engrafted on top of our tuberculosis suppurative infection or something of that kind, and we find quite frequently that infection into the carotid and submaxillary gland comes from the tonsillar region and also from the naso-pharynx.

I think we are at fault very frequently in not making a complete examination of the naso-pharynx, and Doctor Hill has spoken of that, and also of the lingual tonsil region.

This year we have had a number of complications in Denver with the German measles and many of them, I believe, were not measles at all, but were scarlet fever, and frequently we have scarlet fever with the rash disappearing very suddenly, with all the sequelæ of scarlet fever, and the enlarged glands may come from the infection in the throat or from an infection of some other type.

The point Doctor Richards brought out is a very good one, that infection of the pharyngeal maxillary space can quite frequently be found and incised inside with fairly prompt recovery; when incised externally, we have, or we may have, a great many complicating symptoms.

RAY FARQUHARSON, M.B., Toronto, Ont. (closing): I am very grateful to Doctor Nash for bringing to our attention the agranulocytic angina for differential diagnosis. There is no excuse for leaving it out of such a table in diagnosis, except that I just omitted putting it in. It is a condition that, as you all know, is an extremely serious one associated with gangrenous angina of the throat, secondary enlargement of glands high in the neck, and a very low white count.

As far as the sentinel gland is concerned, every once in a while one makes a diagnosis of what is wrong with a patient seriously ill, in whom there is a carcinoma of the stomach, without definite gastric symptoms.

As far as Doctor Richards' branchiogenetic cysts are concerned, I confess complete ignorance of them and am glad to hear of them. I am sure the nose and throat men will think of it long before I have occasion to do so.

In connection with syphilis, occasionally gumma, especially involving the sternomastoid muscle, may be confused with the lymph gland, but it is not a lymph gland enlargement. As to the uveo-carotid tuberculosis to which I referred—our cases have not been published, but there was a fairly complete review of the condition in the *Lancet* during the past year.

I don't wish to give the impression that they recover quickly. Many of them have recovered spontaneously over a long period of time. The first patient that I saw was one who had a tubercular uveitis, following which there occurred a very large swelling of his carotid and submaxillary glands, and in that particular case we had the glands excised (the carotid and submaxillary glands, or part of them), but a number of cases have been reported where they have cleared up spontaneously.

It is important, as Doctor Carmody remarked, to look for complicating infections. The subject is very broad and it is difficult to cover it all.

# THE TREATMENT OF HAY FEVER AND HYPER-ESTHETIC RHINITIS BY IONIZATION

By LEE M. HURD, M.D.

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## INTRODUCTION

Electro-ionic medication or medical ionization, properly called iontophoresis, was much in vogue thirty years ago. Then its use subsided until Warwick reported his successful results. Such ionization is the introduction of ions into the skin or mucous membrane by the galvanic current.

The galvanic current can be produced by several methods, namely:

1. Batteries which give a pure constant current. This set-up has been made for years by different manufacturers. An inexpensive set has just recently been described by Douglas Macfarlan.<sup>1</sup>

2. The galvanic wall plate where the power line is D.C. (direct current).

3. The motor generator.

The report on tests of three apparatus by the Westinghouse laboratory is as follows:

1. *Wappler Wall Plate*.—This device operates from the D.C. supply and the output, therefore, takes on the character of the D.C. from the power mains. Inasmuch as the ripple in the majority of power systems is entirely negligible, the galvanic output of this machine is similar to the output from a battery as regards ripple.

2. *Burdick Galvanic Generator*.—This device consists of a motor generator set, hence the output will have the characteristics of the generator rather than of the current from the power lines. Measurements made on an oscillograph show that the output at milliamperes up to about thirty was almost entirely without ripple, the ripple being so small that it could not be measured with our laboratory equipment.

3. *Another Make of Galvanic Generator*.—This machine also contains a motor generator set with the result that the current delivered has the characteristics of the generator. The generator output has a considerable amount of ripple, the percentage varying with the milliamperage output of the machine. With small amounts of output the percentage ripple is quite small and the percentage

increases when the D.C. milliamperage output increases. Oscillograms made at an output of 30 ma. show a ripple content of approximately 8 percent. The frequency of this ripple is 1,080 cycles per second.

Each type of apparatus has its adherents. It probably makes no difference, as clinically the patients have about the same degree of discomfort, the appearance of the membrane is the same, and so are the after-results.

The current is uni-directional, of low voltage and amperage. The positive pole repels hydrogen, alkalis and metals, and a convenient way to remember this is with the initials "H. A. M. P.," the "P" indicating "positive." The negative pole repels acid, acid radicals and halogens. The galvanic current produces two effects: (1) physiochemical, and (2) physiological. The effects manifest themselves at the point of entry and exit and also along the path of the current between the poles.

The effect at the poles is simple, but what takes place along the path of the current through the body, is still undetermined and subject to much controversy. If the two terminals are metallic plates and are placed against the wet skin, there will be caustic effects, namely, the sodium chlorid of the body fluid will break up into caustic sodium hydroxid at the negative pole, and hydrochlorid acid at the positive pole. To overcome this destructive action, both poles, metallic terminals, must not come in direct contact with the skin or mucous membrane, but must be separated by a pad of cotton, or the electrolyte. The pads must be sufficiently thick so that the caustic chemicals from the metallic poles cannot diffuse to reach the skin and mucosa.

As the current flows through the human body many complex factors come into place besides the action on the sodium chlorid. In the body there are also colloids and other metals and acids. There must be migration, or at least redistribution of ions in the body between the poles, but if the current is kept within physiological limits, there are no demonstrable physiological effects.

#### ELECTRO-IONIC MEDICATION

Iontophoresis or ionization is the attempt to pass certain drugs or chemicals through the skin or mucosa, by means of the direct current. If an aqueous solution is made of a chemical there are positive and negative ions liberated which are moving about in a disorganized way. If a constant current is allowed to flow through



this solution the positive ions go to the negative pole, and the negative ions to the positive pole. For example, in a solution of sodium chlorid the sodium ions go to the negative pole and the chlorin ions to the positive pole. Leduc,<sup>2</sup> thirty years ago, claimed for ionic medication, that it would place the drug where wanted, in any amount, and demonstrated that strychnin sulphate and potassium cyanid could be passed through a rabbit's skin to kill the animal. Pack<sup>3</sup> verified these experiments, but they do not hold for salts of heavy metals.

Subsequent investigators demonstrated that different ions moved at a fixed rate of speed which increased with the voltage and diminished with the distance in the electrolyte through which they traveled. The speed is roughly in proportion to the atomic weight. Hydrogen is the fastest, and the heavier atoms are slower. Therefore, the heavy metals like zinc, copper, tin and cadmium are many times slower than hydrogen. The hydrogen ions being present in great numbers in the body fluids carry nearly all, if not all of the current, while the ions of the heavy metals being very much less numerous, probably do not transmit the current at all.

It has also been demonstrated that the moment the ion of a heavy metal comes in contact with the body, it loses its charge and combines with the proteins, and first forms zinc oxychlorid, then zinc albuminate, the effect taking place beyond the mucosa being due to the passage of the current only. Pack, *et al.*,<sup>3</sup> found that mercury was not absorbed and came to the same conclusion as Turrell,<sup>4</sup> that "the zinc ion attracted from the positive electrode to the negative pole, on arriving at the surface of the body, lags behind, and the current is carried on much faster by the hydrogen ions of the tissues. The zinc ion is thus set free to enter into chemical combination with the tissue constituents and forms an oxychlorid of zinc, which is electrically deposited upon the superficial tissues. The process is one of electrodeposition resembling the electroplating of commerce. The skin or mucous membrane thus forms a virtual negative pole."

This assumes that there is a superficial deposit of zinc combined with the proteins to form the zinc albuminate. Friel<sup>5</sup> says ionization is strictly a local treatment and, therefore, should be applied to the whole area. Lierle and Sage<sup>6</sup> state that "the possibility of the deposition of metallic zinc in living tissue is remote and open to question."

McMahon<sup>7</sup> shows that, "ionizing with zinc sulphate the dog's normal frontal sinus mucosa causes marked changes in the mucosa,

*i.e.*, engorgement of all the capillaries of the subepithelial tissue. There was also present a marked extravasation of red blood cells into the edematous subepithelial tissues, and certain more or less extensive destructive changes in the epithelium." Hollender and Gorin, in a paper presented before the Illinois State Society, May, 1935, state that "nasal ionization, histologically, produces an alterative effect characterized by immediate destructive cellular changes with absence of cilia even after resolution has taken place."

Alden<sup>13</sup> concludes that "clinical observation and microscopic examination of the nasal tissue has thus far shown no evidence of permanent nasal damage following ionization."

My own examination of polyp tissue before and after ionization, showed, with the same dose as McMahon<sup>7</sup> used, some increase in edema to a definite coagulative effect which had fairly well penetrated through the polyp, but no necrosis. It is possible that the difference between McMahon's findings and mine is the difference between normal mucosa and polypoid tissue.

When zinc sulphate is dissolved in water a proportion of the molecules split up into two fragments, namely: (1) an atom of zinc, and (2) a group of atoms of ( $\text{SO}^4$ ). A solution of a substance in water which undergoes this dissociation into fractions is called an electrolyte. The positive and negative fractions of the electrolyte are called ions. Positive ions are called cathons, negative ions are called anions. It has been found that the dissociated molecules in a concentrated solution increase the resistance to the motion of the ions among them, therefore, if one increased the dilution until a point is reached where dissociation is complete, the maximal value of the electrolyte conduction is reached. Therefore, the weaker the solution the better; the strength of zinc sulphate should be 1-2 percent.

Ionization of the nose has a few advocates, Friel,<sup>8</sup> Crabbe,<sup>9</sup> and Franklin,<sup>10</sup> in England, Hollender<sup>11</sup> and Warwick,<sup>12</sup> in the United States. The latter claims much better results by adding a small quantity of tin and cadmium to the zinc, and on account of his claims Alden<sup>13</sup> took up his technic and has reported such favorable results that it has created an interest in this method of treating hay fever and hyperesthetic rhinitis (allergic coryza).

Warwick<sup>12</sup> lays much stress on technical detail, namely, that the electrode must be used only once, the resistance must be low, the electrolyte must be the one which the Burdick Company puts out in ampoules, and that his special generator is the only one which will give the proper current.

There was some criticism that these formulæ were kept secret, but finally a mimeographed formula from the Burdick Company stated that the electrode was composed of zinc 97.1 percent, tin 1.9 percent, and cadmium 1.0 percent. An electrode which I had had analyzed was found to contain zinc 91.8 percent, lead 3.83 percent, tin 2.54 percent, and cadmium 1.70 percent, also a trace of bismuth and iron. This led me to believe that it was made from a crude zinc metal. The present electrode contains the proportion of zinc, tin and cadmium as stated, *viz.*, zinc 86.04 percent, tin 9.48 percent, and cadmium 4.34 percent.

At first the electrolyte was said to contain zinc sulphate grains 9; stannous sulphate 0.9 grains, cadmium sulphate 0.45 grains, and a blue dye. My examination of the contents of the ampoule showed undissolved violet dye and the presence of zinc and cadmium sulphate, and no tin. The present formula has been changed to zinc sulphate 8.92 grains, stannic chlorid 1.42 grains, cadmium chlorid 0.45 grains, to the ounce. My examination of the present electrolyte showed zinc sulphate 7.193 grains, no tin, cadmium 0.374 grain, sodium oxid 0.058 grain, to the ounce. This would lead one to believe that the formula need not be so exact, and is subject to modification and improvement. It is better to have the electrode of the same metals as the salts in the electrolyte, as the acid radical will attack the electrode and form more ions of the same sort.

Remembering that the ions travel with a speed in relation to their atomic weight, zinc being 30, cadmium 48, tin 50, theoretically it would seem that the zinc ions would be deposited much faster than the other two, and considering the much larger proportion of zinc, that the amount of tin and cadmium deposited in the tissues would be very little indeed. How much this is an improvement over straight zinc ionization remains to be determined.

#### TECHNIC

Granting that you have an apparatus to generate galvanic current, whether it is an inexpensive battery set-up or a Burdick machine, it should have meters so that you may know how many ohms, volts and milliamperes you are using.

The Burdick apparatus is especially built for nasal ionization, and eliminates error, as much as can be built into a machine. This has ohm and milliampere meters, terminals which plug in so that you cannot make a mistake as to poles, a clock which times length of current flow and also turns off the current at end of treatment, so that there will be no shock.

According to Warwick, the nose should be anesthetized, then carefully packed with cotton strips saturated with the electrolyte, and the metal electrode, which has been wrapped with cotton, introduced in the center, and more cotton packed around it. The arm cuff separated from the skin by a pad saturated with saline, is placed on the forearm, after the skin has been cleaned with a grease solvent. The negative pole is connected to the arm cuff, and the positive to the electrode in the nose. The ohm meter reading, if satisfactory, should be between 2,000 and 3,000. If higher, Warwick advises repacking the nose, but before doing this to try passing a couple of milliamperes through the circuit and then read again. Usually the skin resistance is diminished and the ohm reading will be lower. According to Ohm's law, amperage equals voltage divided by ohmage; two factors being known, it is easy to obtain the third. For example, ohms being 3,000, milliamperes 10, multiplying ohms by milliamperes gives 30 volts. If 6,000 ohms, it would be 60 volts, so it is well to have the resistance as low as it can be obtained by good contacts at the two poles. Warwick advises a dose of 100 milliampere minutes, that is 10 milliamperes for ten minutes.

This dose is arbitrary as it depends on the surface area of the nose, as well as the time and current. For example, if one nose had an area of forty square centimeters, and received 100 milliampere minutes, another nose of eighty square centimeters would have to have 200 milliampere minutes to receive the same electro-ionic dose.

The current is gradually turned on until it reaches the milliamperage desired. As soon as the current begins to flow at the required strength, the patient notes a metallic taste, and there is an increased flow of secretion from the eyes, nose and mouth.

After the current has flowed for the required length of time it is gradually turned off and the nasal packs removed. The nasal membrane is found to be contracted and coated with a grayish white deposit.

The patient now complains of a headache which will become severe and last for several hours, requiring a sedative. The nose becomes obstructed and the next day the mucosa is covered with a jelly-like fibrin, which persists for from three to five days, during which time there is almost complete nasal obstruction. The fibrin should be allowed to come off spontaneously. The membrane will remain red for some days following.

I have made some modifications in the technic, abandoning the clumsy Burdick cuff, because it was hard to get good contact, and the patients complained of disagreeable sensations in the arm, and



using the old method of the flexible sheet metal, very heavily padded with cotton (one-half inch thick when wet), saturated with normal saline, as the large negative electrode. I administer atropin sulphate, gr.  $\frac{1}{150}$ , before the treatment, in order to reduce the unpleasant salivation, and am now experimenting with the method of flooding the nose with the electrolyte instead of packing, which was very uncomfortable, and in many cases, on account of anatomic irregularities, it was impossible to pack satisfactorily.

Mr. Anderson, president of the Burdick Company, gave me a U-shaped glass tube for this purpose, which I found was devised by J. Sandison Crabbe<sup>9</sup> of Birmingham, England, in 1920. Gale<sup>14</sup> devised a similar tube which was also used in the same way. I have used a simpler and less awkward tube which is made with a hard rubber nasal tip, connected with rubber tubing to a small glass tube just large enough to admit the electrode. Near the end of the glass tube a piece of tape is tied. At the other end of the tape is a two-ounce lead weight. The rubber tubing is three inches and the glass tubing four and one-half inches long. The patient bends forward until the face is horizontal with the floor, the nasal tip is held tightly in the naris by the patient, and the glass tube is brought up vertically beside the patient's ear and held there by the tape which is passed across the back of the head and held in place by the weight. By this method the nose is flooded until the electrolyte runs out of the opposite naris, and the electrode is inserted into the electrolyte in the glass tube. There is much less discomfort, in fact it is a pleasure to the patient compared to packing, and is superior to packing as all mucosal surfaces are in contact with the electrolyte, which is impossible by the pack method. All good things have a drawback, and in this case it is that the olfactory cleft is included, whereas in packing it is not, and the result is an anosmia which remains for some weeks. This defect must be overcome.

## RESULTS

Warwick reports forty cases done over a period of more than a year, with practically 100 percent results, principally for hay fever, a few cases of hyperesthetic rhinitis, and three cases of asthma with hay fever. Many of these have had no recurrence for from one to six years, a wonderful record.

Alden<sup>13</sup> treated nineteen cases in 1933 for autumnal fever during the attack, and all were relieved. Nine had no recurrence the second year, two had similar symptoms as in 1933, eight had such mild symp-

toms that he did not think it necessary to reionize them. In 1934, forty-one cases were ionized prior to symptoms, 50 percent were protected.

The Burdick Company has compiled the results of many men who used the method of Warwick during 1934, and a few cases in 1933. Roughly the cases are as follows:

#### HAY FEVER—CASES REPORTED 521

95-100 percent improved .....	302	58.0 percent
85- 90 percent improved .....	44	8.4 percent
75- 80 percent improved .....	43	8.2 percent
60- 70 percent improved .....	20	3.8 percent
40- 50 percent improved .....	46	8.6 percent
Less than 30 percent improved .....	8	1.5 percent
No relief .....	58	11.1 percent

#### ASTHMA—CASES REPORTED 194

95-100 percent improved .....	76	39.0 percent
80- 90 percent improved .....	8	4.1 percent
70- 80 percent improved .....	17	8.7 percent
60- 70 percent improved .....	9	4.6 percent
50- 60 percent improved .....	25	12.8 percent
40 and under improved .....	5	2.5 percent
No relief .....	54	27.7 percent

#### HYPERESTHETIC RHINITIS—CASES REPORTED 111

95-100 percent improved .....	47	42.3 percent
80- 90 percent improved .....	8	7.2 percent
70- 80 percent improved .....	15	13.5 percent
60- 70 percent improved .....	3	2.7 percent
50- 60 percent improved .....	10	9.0 percent
Less than 40 improved .....	8	7.2 percent
No relief .....	20	18.0 percent

#### ALLERGIC CONJUNCTIVITIS—CASES REPORTED 6

Completely relieved .....	4	No relief .....	2
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#### URTICARIA—CASES REPORTED 22

Completely relieved .....	3	No relief .....	17
Moderately relieved .....	2		

#### MIGRAINE—CASES REPORTED 2

Moderately relieved .....	1	Slightly relieved .....	1
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#### ALLERGIC DERMATITIS—NOT URTICARIA?

2 cases, not relieved

Hollender,<sup>11</sup> using zinc sulphate, reports very much the same results for hyperesthetic rhinitis. Franklin<sup>10</sup> reports hay fever 50 percent, hyperesthetic rhinitis 60 percent, free of symptoms. He uses zinc sulphate 1 percent and gives three treatments using three to five milliamperes for from twelve to twenty minutes.

### CASE REPORTS

A woman, age twenty-seven, has had a watery nose which had become so bad that she could not work on account of constant nasal drip, and sneezing. She was thoroughly tested by two expert allergists with no results, showed a very pale and swollen mucosa with a profuse serous discharge. She was ionized over a year ago, all symptoms are now relieved although the mucosa is still pale. She has since discovered three things which caused her symptoms, pineapple, peas, and something which is in a package called "Spaghetti Dinner."

A man, age twenty-six, for six years had autumnal fever and hay fever, and for the balance of the year sneezing and nasal discharge up to 9.30 a.m. daily. He received protein injections with no result. He was ionized last June and immediately afterward went on a 4,000-mile automobile trip through the Middle West. He has had no allergic symptoms but now has tree pollen coryza.

Another illustration where the mucosa was only wet with electrolyte is the case of a girl, twenty years of age. Nasal obstruction for two years, watery discharge, sneezing, burning sensation in nose and eyes. Many eosinophiles in the discharge, 10 percent in the blood smears. This was one of the first cases done with flooding instead of packing. The technic was poor, most of the electrolyte leaked out and there was none in the nose when the tube was removed. Warwick solution was used on one side, zinc sulphate 2 percent on the other. After-reaction was the same on both sides. Eight weeks later she relapsed. In this case the mucosa was only wet, not covered with the electrolyte.

I am not ready to tabulate my results, as in some cases I have used the Warwick method, in others the Warwick on one side and another technic on the other side, and in others a still different method.

In cases of hay fever ionized during the attack the percentage of good results has been very high; in hyperesthetic rhinitis over 70 percent of the cases have become symptom free.

### CONCLUSIONS

1. The nasal mucosa shows microscopic changes immediately after ionization.
2. Zinc ions do not enter the tissues in any appreciable amount.
3. There is marked clinical reaction of the nasal mucosa lasting about one week.

4. Symptoms of hay fever disappear immediately after ionization in nearly all cases.

5. Hyperesthetic rhinitis is symptomatically relieved in the majority of cases.

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*(For discussion, see page 159)*



## OSTEOMA OF THE NASAL ACCESSORY SINUSES

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Osteomata of the nasal accessory sinuses are not common. The earlier authors writing on the subject agree that the sinus most commonly affected is the maxillary, but later writers give the frontal as the most frequent. The reason is that the frontal was rarely opened. Bony tumors of the sphenoidal sinuses are very rare.<sup>1</sup> Portmann and Retrouvey<sup>2</sup> are cited by Stout as finding only about thirty cases of frontal osteoma on record when they wrote their book on "Cancer of the Nose" in 1927. In 1928, Harris<sup>3</sup> compiled 117 cases including three of his own, and there have been reports of cases by several other authors since that time. Armitage<sup>4</sup> realized the discrepancy in the various estimates of the number of cases, merely cited the figures given by others, and concluded that the condition was a rare one. The great discrepancy between the figures of Portmann and Retrouvey and that of Harris, both of which appeared within a year of each other, is probably due to wide divergence in the standard of diagnosis. Harris has accepted the figure of Gerber<sup>5</sup> made in 1907, and added those cases he found after that date. Beck estimated 180 cases in 1930.<sup>6</sup> Any accurate estimate of the exact number of cases would require a careful check of the reports and tabulation of cases. Such a careful survey might be of real value.

Scudder<sup>7</sup> reports several osteomata of the upper and lower jaws and skull, but only one apparently involved the sinuses (the frontal). Skillern<sup>8</sup> does not mention osteoma by name but mentions stone in the sinuses, which from his description, would seem to be an osteoma.

Most authors found evidence to support the statement that the condition is more common in males than in females, but in the six cases I report all on the left side, three were in males and three in females. However, this group is too small in number to be of value in drawing deductions.

Osteomata of the sinuses have been reported in medical annals for years, although the exact date of the first such report is doubtful. They do not seem to be limited to the human being, as there are reports of their being found in animals by veterinarians.

The removal of such tumors was not very successful and the mortality from attempted removal was very high until the advent of

anesthesia, since which time it has been comparatively low. Diagnosis by the x-ray has markedly increased the number of successful results, as it is of value in determining the extent of involvement and the probable outcome.

Figures indicate that the condition is more common in young people—50 percent of the cases occurring in adolescence and 80 percent before the age of fifty, according to Armitage.<sup>4</sup> This is in a measure supported by the six cases I have to report, in which the ages were 11, 13, 23, 27, 35, and 42 years.

The rarity of the condition is best attested probably, by the fact that no one author has a large number of cases to report. Knapp (cited by Armitage<sup>4</sup>) reported eight cases found in a review of 56,000 records, but these were not necessarily his own cases. A rapid review of the literature indicates that no single writer has reported more than two cases of osteoma of the frontal at one time. My report concerns four cases, and incidentally, although of what significance I cannot state, all have occurred within the past two and a half years.

The etiology of the condition is truly controversial. Harris has reviewed the various opinions on the subject well, and he found that the tumors have been thought to arise from embryonic cartilaginous cells at the junction of the ethmoidal and frontal sinuses (in agreement with Sutton and Scudder), from the periosteum of the sinus walls, from the diploe, from ossified polypi and as the result of syphilis. (Most of the older authors consider syphilis as the main etiological factor but state that recurrence after removal is rare. In none of my cases was the Wassermann positive.) Trauma, of which one of my cases gave a history, sinus inflammation, and tuberculosis are other etiological factors.

Harris<sup>3</sup> also found divergence as to the reports of the site of attachment. He quoted other reports of the site as being: the junction of the frontal sinus and ethmoidal bones far forward; the under and inner wall of the frontal bone and the ethmoid; a pedicle, inseparable from its frontal and ethmoidal origins, continuous with the frontal bone at the inner wall of the sinus; the upper lateral wall in the frontal sinus; the infundibulum; the floor of the frontal sinus external to the duct; the anterior end of the junction of the ethmoidal with the frontal bone; and the septum between the right and left frontal sinuses.

These tumors usually grow slowly, and there is a great divergence in size and involvement. The chief symptoms are external deformity, headache, cerebral paralysis, discharge, and vertigo. All symptoms

are dependent upon the amount and type of involvement. There does not seem to be any definition of the direction of possible extension. In one of my cases the extension was posteriorly into orbit, and in another it was anteriorly with a slight dehiscence in posterior wall in the same case. The external deformity may be in the form of a bulging process, and there may be proptosis of the eye.

The treatment, judging from the results obtained, is undoubtedly surgical. Since no one operator has had a sufficiently large number of cases to evaluate various methods of surgical procedure, the method must depend upon the particular case.

### CASE REPORTS

*Case 1.*—A. G., a white female aged twenty-three years, was first seen on March 9, 1925, because two months previously she began to have headaches and pain over her left cheek, considerable nasal and postnasal secretion, and the left nostril felt obstructed. The family history was not pertinent.

The patient had had measles, mumps, whooping cough, chickenpox and pneumonia. The tonsils and adenoids had been removed, and two months previous the left middle turbinate had been removed. Her appetite was good, but she slept poorly.

The roentgen examination showed slight increased density of right and left maxillary sinuses; right ethmoids were clear, but the left showed slight increased density; the right and left frontals and sphenoids were clear. This indicated infection of right and left maxillary sinuses and left ethmoid. There was an area of increased density in the left upper third molar region with an impacted tooth above. This involved the posterior and inferior parts of the left maxillary sinus. The right and left third molar teeth were impacted.

On March 9, 1925, the left upper third molar tooth and a bony tumor were removed under ether anesthesia, by the regular Caldwell-Luc technic. Two days later, again under ether anesthesia, a drainage tube was inserted in the left side of the neck under the angle of the jaw.

Eleven days later (March 21, 1925), the left maxillary sinus was irrigated and a quantity of broken down blood was obtained. Two months later (June 9, 1925), the discharge was clear, but three months later (September 7, 1925), irrigation fluid contained considerable pus. November 22, 1925, under ether anesthesia, left maxillary sinus operation was performed with injection of alcohol into the left infraorbital nerve, as we were not sure that sufficient pathology was present to account for pain.

One year after the first operation (March 3, 1926), irrigation of the affected maxillaries again returned cloudy fluid. Eight months later (November 13, 1926), the three impacted molar teeth were removed. For the next year and a half she was not troubled, but on March 5, 1928 and again on April 18, 1928, it was necessary to inject the left infraorbital nerve with 50 percent ethel alcohol. After that she developed epileptiform convulsions and was unable to do her work, gradually getting weaker and being confined to hospital for several months before she died on May 10, 1930.

Autopsy failed to give a satisfactory anatomical or pathological diagnosis.

*Case 2.*—R. G., a white male aged eleven years, was first seen on August 11, 1925. The father and brother were living and well, but the mother had pernicious anemia. The patient had had measles, mumps, pneumonia, "flu" and tonsillitis, and four and one-half years previous a swelling of the left cheek had been noticed. At operation six months later, a mass of bone was removed from the region of the left maxilla. One year previous to the first consultation, deformity of the teeth was noticed and there was an increase in the size of the alveolar process.

On August 12, 1925, under ether anesthesia, the left maxilla was opened and the growth removed. No cavity that could be designated as a maxillary sinus was found. The pathologists report was: "Numerous small pieces of bone occupying the bulk of a hen's egg. Sections show that many of the pieces consist of normal bone with no canals, only a few clefts. No evidence of malignancy was found."

Diagnosis: Osteoma.

*Case 3.*—P. G. P., when first seen on March 8, 1932, a white male twenty-seven years old had suffered night and day for three weeks with continual headaches. The family history was negative. The patient had had a tonsillectomy in 1928, a tonsil tag removed in 1930. For three years he had been troubled with occasional frontal headaches which had recently become more frequent and more severe. They were accompanied by considerable post-nasal discharge. Formerly the pain was more severe in the mornings, latterly the intensity did not vary. His general health and his appetite were good and he slept well.

*Examination:* Mouth.—The patient had had considerable dental work. There was a tonsil tag on the left attached low on the posterior pillar. The posterior pharyngeal mucosa was inflamed, with granular hypertrophy. There was a large amount of thick yellow discharge in the nasopharynx and the mucosa of the palate was congested and swollen.

Nose.—The septum was straight. There was marked congestion of the right inferior and middle turbinates with thin mucopus and obstruction. On the left, the middle turbinate was inflamed, but there was no polypoid degeneration and no free pus.

Sinuses.—There was marked tenderness over the left frontal and supra-orbital ridge. Transillumination on April 1, 1932 gave these results: Maxillary sinuses, left III, right II, frontals I.

X-ray examination on March 24, 1932 showed the right frontal sinus to be normal, the left frontal was continuous with the left anterior ethmoid. There was a tumor mass occupying the space between the upper portion of the anterior ethmoid and the lower portion of the left frontal. There was no evidence of disease of the maxillaries or other sinuses.

On April 2, 1932, under gas-ether anesthesia, the external plate over the lower part of the frontal was removed and a bony tumor about the size of a small olive was revealed. There was considerable pus retained above the tumor with polypoid degeneration of the frontal mucosa. The tumor was apparently not attached and was easily removed. This revealed an intact inner plate. Much of the superior orbital wall was taken away and the anterior part of the middle turbinate was removed and the nasofrontal duct was enlarged.



Eleven days after operation (April 13, 1932), the roentgen examination showed some cloudiness of the left frontal sinus, but no evidence of bony growth.

The diagnosis was osteoma of the left frontal sinus with retention.

Subsequent history, as given in letter of May 6, 1935, from patient: "After the operation you performed there was a complete cessation of headaches until in the winter and spring of 1934 when I began to have dull headaches in the frontal region—mostly bilateral but more severe on the left side. Accompanying this was rather severe nasal congestion. X-rays were taken at Passavant Memorial Hospital, Chicago, but nothing definite was determined except sclerotic bone changes.

"About the first of November, 1934, I had more severe headaches, throbbing pain around the left eye and considerable circumorbital edema, enough at least to make it look definitely puffy.

"X-rays were again taken—this time at Washington Boulevard Hospital. From the finding of the lateral views especially, we all thought that there had been a recurrence of the tumor. So the latter part of November Dr. Lynn F. McBride again operated. To the surprise of everyone no tumor was found—the sinus wall was smooth and well healed. He could not, however, find any sign of a nasofrontal duct or artificial opening so apparently the sinus was completely closed.

"Doctor McBride went in slightly above your incision. I was in the hospital for three or four days—recovered quite rapidly, the edema went down in about ten to fourteen days and today one has to look very closely to detect either scar. Doctor McBride made a rather large opening into the sinus, which has remained open very well.

"I've had only one or two slight headaches since November."

*Case 4.*—R. B. K., on April 13, 1932 a white male thirty-five years old, was referred to me by Dr. F. O. Kettlekamp of Colorado Springs. His chief complaints were a recently developed hesitancy in speech, partial paralysis of the right arm and leg, and, upon leaning the head well forward and down and then rising to an erect position, a sensation as of fluid moving in his head. About three years previous he blew his nose exceptionally hard, and a swelling of the left eye resulted immediately. He consulted a physician at once. The latter recommended wet witch hazel dressings, and in about three or four days the swelling had subsided. For two or three years he had suffered from headaches which were rather severe, but not frequent. These started in the frontal region and extended all over his head. When they were severe things turned black before his eyes. He had had none since November, 1931 (five months previous). By occupation, the patient was a salesman, and on January 1, 1932, he noticed while with a customer that he was unable to talk. He went home at once and soon recovered. The next day he noticed a slight lameness of the right arm which continued to grow worse. Ophthalmologic examination showed no visual disturbance whatsoever, although there was slight proptosis of the left eye downward and outward.

*Examination:* Throat.—There was an infected tonsil tag on the left, scar tissue contraction on both sides of the palate, the left side of the tongue showed slight apparent atrophy, the tip of the tongue seemed to deviate slightly to the right. There was no palsy of the soft palate.

Palpation of the rim of the left orbit revealed on the upper rim a tip of bone about one-half inch from the inner canthus. There was a question of actual dehiscence.

The neurological examination revealed no cranial nerve paralysis. The reflexes were normal except that the right kneejerk could not be elicited. There was partial paralysis of the right extremities and wasting of muscles of arm and leg, hesitation in speaking and sometimes motor aphasia.

The roentgen examination showed a large, irregular, bony tumor almost completely filling the left frontal sinus. This tumor had destroyed the roof of the orbit by pressure necrosis and pushing its way backward through the posterior wall of the left frontal sinus projected into the cranial cavity. There was a dumbbell shaped area of apparent rarification of the skull extending from the frontal sinus under the left parietal bone. This was thought to be air which was caught between the dura and the calvarium.

On April 16, 1932, under ether anesthesia, incision was made below the left supraorbital ridge and perpendicularly to give more room. The plates of the frontal sinus were removed and the tumor exposed. An irregular spine was found extending into the orbit, the roof of which was destroyed. Practically all the anterior portion of the internal wall of the frontal sinus was removed and the tumor pried loose. This was followed by a gush of blood and cerebrospinal fluid, which escaped from an opening about the size of a dime in the dura, and also of air or gas. The edges of this opening appeared purulent and necrotic, and it apparently led into a smooth-walled cavity which was either the remnant of a large empty brain abscess or the lateral ventricle. The cavity was packed with about twelve inches of one and one-half inch iodoform gauze. The vertical incision over the left frontal was closed with horse hair sutures. The outer angle of the incision below the brow was closed with skin clips.

The pathologist's report was: "Section of pieces of bone the size of a pea showed osteoma. Later tissue and bone of about the size of a cherry was also sent to laboratory. The sections showed dense, compact bone containing only a few small Haversian canals. There was no evidence of malignancy." The diagnosis was: Osteoma.

The patient made an uneventful recovery except for a moderate seepage of cerebrospinal fluid from the wound, and a diplopia resulting from removal of the superior oblique muscle pulley. This disappeared.

On November 11, 1932 (seven months after operation), the patient had a convulsion which followed swimming and another attack six months later also after swimming. He was advised not to allow fluid in the nose. He was seen by Dr. Joseph C. Beck who concurred in advice.

Patient was seen in June, 1933 and had completely recovered even the muscles of the extremities and speech.

On January 20, 1935, he suffered another attack, which he describes as follows:

"It came on me very much the same as the others, that is, with no warning and affected me very much the same. My right hand and arm started to shake and become stiff, my head commenced to jerk around to the right, my right leg started to straighten and jerk, I fell from the chair upon which I was sitting to the floor and that is the last I remember. I was 'out' probably

between thirty and forty-five minutes. Both immediately before and for sometime after (an hour or so) I could not talk at all."

Perfectly normal since this time and continues with his work.

*Case 5.*—V. S. M., a white girl, aged thirteen years, noticed a lump on her forehead about one month previous to July 25, 1934. Roentgen examination of the sinuses on July 16, 1934 showed the frontals fairly large. There was a slight density of the left maxillary sinus and right and left ethmoids which indicated slight infection of those sinuses. There was a moderate sized opaque substance, probably osteoma, in the frontal sinuses slightly to the left of the midline, and it had apparently broken through the anterior frontal plate.

On July 25, 1934, under ether anesthesia, the tumor was removed from the left frontal sinus, both frontals were opened and as septum was involved and part of growth found in right sinus, the membrane was removed in its entirety.

Recovery was uneventful and complete.

*Case 6.*—B. H., a white woman, aged forty-two years, was referred to me by Dr. Robert Packard on June 23, 1934. She had had sinus trouble ever since five years of age, at eleven years she remembered having a very bad cold and a broken septum, but her memory was not good.

Roentgen examination was made of the sinuses. The frontals were of average size. There was an area of increased density practically filling the left frontal sinus which had the appearance of an osteoma. It was apparently attached to the posterior wall. There was slight increased density of the right and left ethmoids and the right and left maxillary sinuses suggesting a moderate infection of those sinuses. There was no evidence of retention.

On September 1, 1934, under avertin anesthesia the tumor was removed from the left frontal sinus and was found attached to frontal septum.

Three months later the patient reported her memory was much improved and there was a lessening of the nervousness.

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## DISCUSSION

WILLIAM B. CHAMBERLIN, M.D., Cleveland, Ohio: To go over this situation briefly, there are apparently five theories as to the origin of osteoma of the frontal and ethmoid sinuses, each of which has its proponents and enthusiasts, and each of which is vigorously opposed by the champion of his own particular theory.

(1) That they originate from fetal remains of cartilaginous tissue, the ethmoid being laid down in cartilaginous tissue, the frontal in connective tissue. Against this theory is the fact that no remains of cartilaginous tissue have ever been found in the osteomata which have been removed. This theory is championed by Arnold, Rokitansky, and Boenninghaus, who see a parallel between osteomas and exostoses of the long bones of the limbs, which usually occur at the epiphyses.

(2) The origin in the periosteum is vigorously supported by the French school, notably Sappoy and Dalveau, as well as by Bornhaupt, Tauber, and Zuckerkandl.

(3) The diploic origin. In support of this Chapman reports a case with a small pedicle arising from the diploe above the frontal sinus.

(4) Others advocate the possibility of suppuration within the frontal sinus as being a causative factor. This doesn't seem to me to hold because I think the suppuration was secondary and due to retained pus and blocking.

(5) Trauma plays an important part.

If one can draw any conclusions from the literature, osteoma of the frontal and ethmoid sinuses must be a fairly rare disease or anomaly. Gerber in 1907 found only eighty-seven reported cases. To these Harris in 1928 added twenty-eight others, two of them being his own. Armitage reports them as occurring more frequently in males than in females, and 80 percent occur before the age of fifty. By far the most frequent incidence is in young people during the second and third decades of life. This is the age, of course, during which trauma is most likely to occur.

JOSEPH C. BECK, M.D., Chicago, Ill.: In the few years I have been in practice, I have had just four cases of osteoma of any considerable size. That doesn't mean exostosis or osteogenesis, or anything like that, just true, proven, histologic cases of osteoma. They have a real histologic picture, histologic appearance.

The traumatic theory holds well and it is the nearest approach because almost everybody gets a bump at some time or other in his life, but you can't call that an osteoma.

I want to speak about a practical application which may be useful if this happens to you. In a case that I have had of an osteoma that has come under my observation since I made my report in reviewing Denker and Kahler's record of



the osteomata that have been collected the world over, there was a point in regard to a condition that fooled me. The x-ray picture fooled me. If I had taken a lateral view, as should be done in these cases, I wouldn't have been fooled, and if I had listened to the radiologist, I wouldn't have been fooled, but I went ahead, operating on an osteoma of the frontal sinus, being sure there was a perfectly normal posterior wall of the frontal sinus and the space between the tumor and the posterior wall. I went down and took away part of the dura that was involved in the tumor and had a brain exposure of an inch and a quarter in size. There was no evidence of any suppuration present.

I was fearful of a secondary meningitis. The sphenoid tumors, osteomata, that is, are inoperable. It is better to leave them alone than have the patient develop a meningitis. That is the consensus of opinion on tumors, and yet we have heard the report of a clearly removed osteoma of the sphenoid in this discussion.

Now, in this man I immediately covered the frontal lobe, by taking a big piece of fascia lata, and I laid it over this exposed brain, put over it a piece of gutta percha, and on top I put iodoform gauze, and four days later I removed this pack and did the final closure. Through this I might have gotten into trouble with a foreign country, because he was a foreigner, from a South American country, and, moreover, he was a vice-president. I am not bragging about the kind of patient, but he was vice-president of one of those republics down there and you can imagine how I felt when his brain was exposed.

# BENIGN LARYNGEAL LESIONS PRODUCING HOARSENESS

(DIAGNOSIS AND TREATMENT)

By CHEVALIER JACKSON, M.D.

Philadelphia, Pa.

*Definition.*—*Hoarseness* is a general term applied to a rough quality of voice. The pitch is usually lowered at the same time. *Huskiness* is often used synonymously but is more correctly applied to the more severe degrees. *Raucity* or *raucousness* used in adjectival form of raucous voice is usually understood to be a loud as well as hoarse voice, though the noun *raucus* is the literal Latin translation of hoarse. "*Strident*" conveys the idea of shrillness as well as loudness, while *stridor* is applied to a harsh shrill respiratory sound independent of phonation. "*Croupy*" is applied to a quality of cough rather than of voice, but it is usually accompanied by hoarseness. The word "benign" is used in the accepted medical sense of distinction from malignant growths. Many non-malignant conditions are fatal hence not benign in the literal sense of the word. All abnormal laryngeal conditions should tentatively be regarded as malignant and serious, until conclusively proven benign.

## THE MECHANISM OF HOARSENESS

For the production of a tone free from hoarseness it is necessary for the vocal cords to (1) approximate; (2) draw tense; and (3) vibrate. Anything that interferes with any one of these three mechanical movements will impair the quality of the note and this impairment constitutes the symptom we know as hoarseness.

*Approximation* may be interfered with by: (1) tumor or other tissue mass between the cords; (2) feebleness; (3) paralysis; (4) fixation or limitation of excursion of the cricoarytenoid articulation.

*Tension* may be interfered with by: (1) paralysis; (2) feebleness; (3) fixation.

*Vibration* may be interfered with by thickening (inflammatory, neoplastic), neoplasm or inflammatory tumor acting as a damper, feeble tension, incomplete approximation.

## PATHOLOGY

Omitting entirely the neurologic conditions producing hoarseness, and disregarding overlapping, the local laryngeal conditions may, for convenience of consideration, be grouped as follows:

Benign growths of inflammatory origin: edematous polypi, vocal nodules.	Feeble tensor muscles.
Hematoma, organizing.	Feeble adductor muscles.
Cysts.	Trauma.
Benign neoplasms.	High tracheotomy.
Scleroma.	Foreign body, larynx, trachea, pharynx, hypopharynx, esophagus.
Endotracheal goiter.	Laryngospasm.
Systemic diseases.	Vocal abuse.
Leprosy.	Contact ulcer.
Acromegaly.	Perichondritis.
Nephritis.	Chondral necrosis.
Lichen.	Cricoarytenoid arthritis.
Pemphigus.	Cricoarytenoid ankylosis.
Herpes.	Cricothyroid ankylosis.
Angioneurotic edema.	Cicatrices.
Tuberculosis.	Stenosis, (a) congenital,
Lupus.	(b) acquired.
Syphilis.	Perichondritis.
Pachydermia.	Phonation with ventricular bands or other folds.
Pachylaryngitis.	Chronic edema.
Keratosis.	Non-inflammatory edema, chronic.
Leucoplakia.	Acute edema.
Eversion of the venticular wall.	Infections.
Prolapse of the sacculus.	Mycosis, blastomycosis, actinomycosis.
Anomaly.	Fibrinous corditis.
Dislocation, (a) thyroid, (b) cricoarytenoid, (c) cricothyroid.	Chronic laryngitis, infiltrative, hypertrophic, atrophic, sicca, subglottic, et cetera.
Laryngomyopathy, myositis, et cetera.	
Myasthenia laryngis.	

*In children* in addition to many of the foregoing possibilities we must consider:

Diphtheria.	Indrawing of the upper laryngeal aperture.
Measles.	Rickets.
Infective laryngotracheitis.	Deficient cartilage or stiffness of cartilage.
Non-membranous croup.	Malformation.
Scarlet fever.	Mutation.
Screamers' nodes.	
Arrest of development.	
Congenital webs.	

In the *newborn* we must consider additionally :

Trauma to larynx (forceps delivery).	Anomalies, malformations.
Trauma to recurrent or vagus (forceps).	Webbs, and other anomalies.
	Congenital papilloma.

*Benign Tumors.*—The following list shows the benign tumors encountered in personal experience and includes both inflammatory tumors and benign neoplasms :

Granuloma.	Lymphoma.
Hematoma, organizing.	Myoma.
Edematous polyp.	Lipoma.
Tophi.	Chondroma.
Angiona.	Xanthoma.
Adenoma.	Osteoma.
Papilloma.	Amyloid tumor.
Fibroma.	Thyroid tumor.

Obviously it is impossible within the necessary limits here set for us to consider adequately even one of these many local laryngeal causes of benign hoarseness. We must confine ourselves to the general consideration of methods of diagnosis and treatment.

#### DIAGNOSIS

Perhaps the most frequent and certainly the most deplorable error in diagnosis is to mistake a malignant for a benign condition. In adults, then, it is a safe rule to make a working diagnosis of malignant disease in every case of hoarseness, and to work backward by exclusion. And it is not only as a primary procedure that this is necessary ; it should be repeated from time to time throughout the course of management of any chronic laryngeal disease. Over and over again a primarily benign morbid condition in the larynx has become the site of malignant disease. Over and over again there have come in patients from whose larynx specimens taken elsewhere have been found negative, yet biopsy has revealed a cancerous process. Over and over again the locality of excision of a lesion of unquestionably benign history has become the site of a malignant process. This does not mean that the previous biopsy has caused malignant change ; nor does it mean that there was an overlooked malignant process previously present. Nearly thirty years ago I made the statement here in Toronto at a meeting of the British Medical Association that malignant disease rarely develops in a perfectly normal larynx.<sup>2</sup> This was questioned by the highest authority of the time ;<sup>12</sup> but the observation has been abundantly corroborated by hundreds of cases since. Clinical



experience in dealing with cancer elsewhere on human surfaces shows that malignant processes are prone to localize on the site of chronic disease; so that analogy supplies no reason to question the possibility of a cancerous process developing at the site of a chronic benign lesion. Be this as it may, clinical facts render it advisable in treating chronic laryngeal disease such as recurrent papillomata, chronic hypertrophic laryngitis, contact ulcer, or any other chronic process, not to rest content with the benign character of the initial diagnosis, but to regard eternal diagnostic vigilance as our first duty to our patient.

In the communication above referred to there was mentioned the case of a man with advanced endothelioma of the larynx who had been treated for chronic laryngitis for ten years. The advanced state of the growth when the patient came indicated that malignant developments were of nearly two years standing. Curiously, twenty-four years later the son of that patient came in with the history of treatment elsewhere for chronic laryngitis for ten years, and on biopsy the condition proved to be malignant, though the son's laryngeal disease proved to be a squamous celled carcinoma. Both of these patients died of recurrence, two and one-half years and one year, respectively, after laryngectomy; both had lost their chance of cure by laryngofissure because of a late diagnosis and both of them illustrate the importance of eternal vigilance in early detection of malignant developments while the patient is under treatment for a benign disease causing hoarseness. Obviously malignant disease had not been present for ten years in either case.

One of the most common causes of failure to make a diagnosis is the limitations of the laryngeal mirror. Invaluable and indispensable as this instrument is we must admit the fact that there are some regions of the larynx invisible in it in all patients and other regions invisible in it in some patients. The inadequacies of the laryngeal mirror may be enumerated as follows: 1. The ventricular floor, walls and roof cannot be seen. 2. The subglottic region posteriorly is hidden from view. 3. In many patients the anterior commissure is hidden under a pendulous epiglottis. 4. In children under about five years of age no part of the larynx can be seen in the mirror.

For the complete diagnostic visualization of the larynx, then, it is clear that direct laryngoscopy is required. This does not, however, lessen the importance of, or necessity for, mirror examination except in case of children too young to cooperate in the use of the laryngeal mirror.

The differential diagnosis as among the many diagnostic possibilities requires something more than mere inspection. The following

routine has stood the test of time. All the steps may not be necessary in every case, but in no instance has there been a failure to make a diagnosis when all or a sufficient number of these measures have been carried out.

- |   |  |
|---|--|
| 1. Anamnesis.   | 7. Serologic tests.  |
| 2. Laryngoscopy, indirect.  | 8. Blood examination.  |
| 3. External palpation.  | 9. Systemic test, therapeutic, with mercury (not potassium iodid). |
| 4. Laryngoscopy, direct.  | 10. Biopsy.  |
| 5. Complete, thorough, general examination, chest, heart, lungs, neck, reflexes, etc. | 11. Bacteriology.  |
| 6. Roentgen-ray examination of chest and neck.  | 12. Bronchoscopy.  |
|   | 13. Esophagoscopy.   |

In the differential diagnostic consideration of tuberculosis, syphilis, hemorrhage and benign and malignant growths as a cause of hoarseness the following points are to be remembered:

1. The lesion may not be in the larynx but may be in the central nervous system or anywhere between the centers and the larynx in the course of the vagi or the recurrent nerves.
2. Tuberculosis or syphilis may coexist in the same lesion with cancer or all three may be combined in one mixed lesion.
3. In differential diagnosis little dependence can be placed on deduction or inference.
4. A syphilitic lesion in the larynx inefficiently treated may remain stationary for years.
5. A positive serologic test does not necessarily mean that the lesion in the larynx is syphilitic.
6. Only biopsy can determine the diagnosis and prompt resort to it is always in order; moreover, if the condition is malignant we must have a specimen of tissue to determine the degree of malignancy.

#### ETIOLOGY

Knowledge of the cause, the underlying, essential cause considered apart from etiologic mechanism, is essential for best results in rational, efficient treatment and prophylaxis.

The cause of true *neoplasms* is unknown. Etiology of the tumors of inflammatory origin is involved in the manifold meshes of the network of causes of chronic laryngitis. *Hematoma* and *organizing hematoma* are not primarily inflammatory. The cause of hematoma is usually violent abuse of the voice as in singing, vehement lecturing, shouting, vociferous cheering at football games and the like. The hematoma does not become absorbed as a rule. If not ruptured

instrumentally it usually becomes organized into a small, firm tumor that will remain permanently unless removed. This epithelialized, organizing hematoma is one of the commonest of benign laryngeal tumors, and is a common cause of hoarseness. It is not a true angioma, histologically.

*Eversion of the ventriculus or prolapse of the sacculus laryngis* may be caused by inflammatory changes but often it seems to be a soft-tissue dislocation of unknown origin.

*Dysphonia Plicae Ventricularis*.<sup>9</sup>—Phonation with the ventricular bands is a frequent cause of hoarseness yet it is not noted in the text-books. It occurs in three forms: 1, the usurpative, in which an aggressively active pair of ventricular bands rush together and vibrate before the true cords have time to function; 2, the vicarious, in which the ventricular bands assume the duties of phonation because of the absence of one or both vocal cords; and, 3, the vicarious in which the true vocal cords are propped apart and damped by a tumor or other pathologic tissue between the cords.

*Inflammatory Diseases of the Larynx*.—The causes of inflammatory diseases of the larynx, including tumors of inflammatory origin are:

Vocal abuse.	Tracheobronchitis.
Tobacco.	Cough.
Alcohol.	Trauma.
Dusty atmosphere.	Hammer and anvil, as in contact
Air borne infection.	ulcer.
Focal infections; lymphoid tissue,	Medication, local, general.
tonsils, faucial and lingual; nasal	
accessory sinuses.	

*Vocal Abuse*.—Unquestionably the greatest of all causes of laryngeal disease is the excessive use of its normal function. The patient with chronic laryngeal disease is almost always a person who either talks constantly or uses his voice professionally, or, often, both. There is little use asking the patient the question. For some curious reason a patient who talks all the time he is awake will insist he talks very little. It is not only the singer, the lecturer, and the huckster who abuse the larynx; teachers are especially frequent sufferers of the larynx. Persons who talk in noisy factories often develop chronic hoarseness. The noise incidental to our modern life is a large factor in the great and increasing incidence of hoarseness and laryngeal disease. To talk in a subway train is utter ruin to the larynx; railroad trains, omnibuses or automobiles are almost as bad. Certainly to converse in the average city street involves abuse of the larynx.

Talking against a noise, that is to say to force the larynx to produce a tone loud enough to be heard above a noise, is certain sooner or later to result in an impaired quality of tone and, in most persons, hoarseness. There is a great variation in the amount of abuse the larynx of different individuals will stand; but every larynx has its limit. To go beyond this limit means thickening of the cords, and a thickened cord means a hoarse voice. Not only is a thickened cord a poor vibrator but it throws great additional work upon the tensor muscles. These muscles instead of growing stronger grow more and more feeble, less and less able to cope with the increased requirements. A vicious circle is established, and this renders cure a long and tedious process even with a cooperative patient. Public speaking is a common form of vocal abuse. In a hall with poor acoustic qualities the abuse is intense. Speaking out-of-doors is the worst of all forms of vocal abuse. The speaker does not get the reflection of his voice back to him; though he does not realize that this is the trouble, he feels that he is not reaching his hearers forcibly enough and instinctively he puts more and more force into his phonation. Hoarseness follows and if he persists in frequent outdoor speaking engagements, chronic hoarseness will persist for life unless a long and faithful adherence to a silent régime is maintained.

Of course in some instances the vocal abuse is at least partly incidental to the disease itself as for instance when a tumor on the cords demands the use of great force to produce a tone. In these cases there is also increased work for the tensor muscles and they may give out. Then myasthenia laryngis is added to the mechanical tumoral difficulty, and to the chronic engorgement secondary to the constantly forceful use of the larynx.

Some patients have the "throaty" voice that involves vocal abuse incidental to every word they say. Other patients have not only this type of voice but they habitually talk too loudly. In fact in this country loudness of voice is almost universal, even in drawing rooms and at various social functions. A large social dinner should not be a noisy affair yet talking against the general hum of conversation, along with breathing the atmosphere laden with tobacco smoke, renders such a function a common cause of hoarseness.

A common form of vocal abuse is *shouting and cheering* as at baseball and football games. Enthusiasm is high and being out of doors the violence of the phonatory efforts is not realized. In this form of vocal abuse the hoarseness may result from vascular engorgement, arthritis or myositis from injury to the joints or musculature, or more often from *hematoma* on the cords. The hematoma may be



absorbed or it may rupture; usually it becomes organized and the hoarseness remains permanently unless the growth is removed instrumentally.

*Tobacco.*—Contrary to that of alcohol the effect of *tobacco* in producing chronic disease of the larynx is local not systemic. It is not the nicotine but the empyreumatic oil produced by the destructive distillation of the burning tobacco that causes the injury to the laryngeal mucosa. This injurious effect is not confined to the smoker himself but is noticeable in the larynx of anyone who remains long in a room whose atmosphere is saturated with tobacco smoke. Chewing tobacco, which used to be so common, is little if at all injurious locally; if injurious at all it is only by the general depressing effect; this is usually more noticeable elsewhere in the body than in the larynx.

*Alcohol.*—As a clinical fact it has long been known that *alcohol* is a potent cause of laryngeal disease acute and chronic. It seems to be the impression that local irritation of alcoholic contact is the mechanism. This is a mistake; alcoholic beverages do not reach the larynx. Observation in thousands of patients justifies the opinion that it is the prolonged peripheral vasodilator effect of alcohol that is so injurious to the laryngeal mucosa. The mucosa of the larynx of the chronic alcoholic that used to be so common before the days of prohibition was always that of an engorged larynx. It was evidently the chronic engorgement of the cords that was responsible for the hoarseness. The engorgement is noted within twenty minutes of the taking of a single drink of whiskey, but if not repeated the engorgement subsides within a few hours. It is the constant reptition of the alcohol that results in the vasomotor condition resembling paralysis and chronic engorgement. This "whiskey voice" is a form of hoarseness that disappeared from the United States during the prohibition period. At the present rate of advertising of the liquor interests the development of a new group of addicts to chronic alcoholism is only a question of time.

*Medication Producing Hoarseness.*—Silver nitrate locally applied is a common cause of hoarseness and the damage done by prolonged applications even of dilute solutions may render the patient hoarse for life. The use of very strong solutions for diphtheria in the days before antitoxin caused life-long hoarseness in the few children who survived. Of the internal medicaments potassium iodid is the commonest as a cause of hoarseness. Its selective effect on the larynx may go so far as to produce acute edema of the larynx, or a chronic engorgement; either of these conditions is associated with hoarseness.

## TREATMENT

Search for and elimination of the cause is fundamental to the treatment of all kinds of hoarseness of which the etiology is known.

The first step in the treatment of almost all forms of benign disease of the larynx is rest of the larynx. If we do not stop the *vocal abuse* of the constant talker or the professional user he will utterly defeat any remedial measures we may undertake. This is most important in the treatment of the inflammatory diseases such as *contact ulcer* but applies with almost equal force to pachydermia, vocal nodules, chronic laryngitis, inflammatory tumor-like formations and even benign true neoplasms. A régime of silence for a few months is to be followed by gradual resumption of very moderate use of the voice. During the silent period twenty-five to fifty words may be allowed daily but they must be spoken in a low tone in a quiet place. By low is meant low as to loudness, not pitch. After a few months the number of words allowed may be increased gradually, but a rigid control must be kept. The patient must never talk in a noisy place, such as in an automobile, subway car, omnibus, railroad train or even in a city street.

Absolute silence should be enforced during an attack of acute laryngitis such as almost every one gets every winter when such infections are prevalent. A professional vocalist or speaker is usually very difficult to control in this matter. A certain pride together with fear of rumors of impaired voice leads many to go through with their duties when the larynx is acutely inflamed. Silence at such times is both a prophylactic and a therapeutic measure of the utmost importance.

*Tobacco smoke* should be eliminated. If the smoker is ordered to quit smoking he will soon either cease to take any advice or treatment, or start on a search for a physician who will permit smoking; in either case he will not get well. A better plan is to allow him, say, four cigars daily with the understanding that he is to mark off the distal three centimeters of the cigar before he lights it; he is to throw the cigar away as soon as the mark is reached. By this plan he gets very little of the empyreumatic oil of tobacco, not only because very little tobacco is consumed but because the long, proximal, cool four-fifths of the cigar serve as a filtering condenser that removes much of the laryngeally irritating oil. Similar instructions apply to cigarette smoking. About fifteen millimeters, say the breadth of a finger nail of the distal end of the cigarette is marked off with a lead pencil before igniting it. It requires courage to throw the cigarette away when the

mark is reached, but when the physics and chemistry are explained and the alternative of total abstinence from smoking is impressed, most patients cooperate; whereas, if totally forbidden cigarettes most patients after a time will fall back into the old vice of perpetual smoking, and will lose the benefit of the cooling, condensation and removal of the empyreumatic oil. The smoker of a pipe should take a fresh one every time and the stem should be long. The old churchwarden clay pipe was ideal from the laryngologic viewpoint. The hookah is even better.

*Nasal breathing*, if impaired, should be restored to obtain the proper warming, moistening and filtering of the inspired air before it reaches the larynx. Moreover, good, clear phonation requires the aid of nasal resonance.

*Chronic Foci of Suppuration*.—Focal infective disease in the nasal accessory sinuses, the lymphoid tissue in the fauces, nasopharynx or lingual region, must be eliminated for prophylaxis as well as treatment.<sup>14</sup> The dripping of pus into the laryngopharynx is a menace to the laryngeal mucosa. Probably the lymphatics are channels by which the infections reach the larynx.<sup>15</sup>

The local treatment of inflammatory laryngeal disease is voluminously considered in the standard textbooks, and the advice therein given may be followed except in the mistaken advocacy of the local use of silver nitrate. This form of medication is one of the commonest causes of chronic hoarseness.

*Benign tumors* require removal by direct laryngoscopy. The removal of the tumors of inflammatory origin such as vocal nodules, edematous polyp, granuloma, require superficial removal flush with the surface. It cannot be too emphatically stated that excision of normal basic tissue is a mistake. *Remove no normal tissue* is a cardinal rule in benign conditions. In recent *hematoma* simple rupture is all that is necessary to forestall permanent tumor formation. Organized or *organizing hematoma* requires only superficial removal. In dealing with benign neoplasms and tumor-like formations on the vocal cord it is always necessary to remember that a convex edge is better for phonation than a concave edge; if normal tissue be taken away a concave edge and damaged voice will result. The fact that the growth is benign must be always in mind. Benign growths do not justify the radical measures proper for malignant disease. They repullulate on the surface, they do not infiltrate, hence removal of normal base is needless. Anyway recurrence of a benign growth is no disaster. In many sad cases a patient has come to us for restora-

tion of voice irretrievably lost because radical removal of a benign growth has left a concave edge to the cord. The tensors cannot bring the postoperative edge into perfect apposition.

In dealing with *multiple papillomata* we encounter the unique situation in tumors that they appear in crops in totally new locations; radical extirpation in one location will have no deterrent effect on new papillomas appearing in new locations.<sup>12</sup> The best plan is superficial removals repeated often enough to prevent the development of obstructive dyspnea. Multiple papillomata in children constitute a benign, often self-limited disease.<sup>8, 12</sup> If radium be used the radiologist must beware of causing perichondritis, chondral necrosis and laryngeal stenosis; the cartilages of the larynx are exquisitely sensitive to radiation. In our early cases of roentgen-ray treatment of multiple papillomata most distressing cases of stenotic perichondritis resulted. The roentgenologist of today has better control over the ray and may be trusted to avoid these dire consequences.

*Endotracheal goitre* usually is involved in a malignant process when it comes under observation but in any case requires extirpation; if benign this should be done endoscopically.

*Cysts* call for endoscopic removal. Deep radical extirpation is not necessary but all of the cystic wall should be removed. To take off every vestige of wall and yet take out no normal tissue requires operative precision.

*Myasthenia laryngis*.—Weakness of the intrinsic muscles of the larynx is a common cause of hoarseness. It most often affects the tensor muscles. Feebleness of the tensors is usually not a neurologic but a muscular condition. The chief cause is vocal abuse. A predisposing cause is general physical weakness or fatigue. It is peculiar to myasthenia laryngis that, in these cases increased work and exercise do not produce increased muscular strength and endurance in the way they do in the general muscular system. This is the factor that the vocal teachers and even laryngologists do not seem to understand. The vocal teacher is always seeking some new method of vocal exercise, when, in reality, what those intrinsic muscles need is rest. About twenty-five words a day may be allowed if spoken in a low tone in a quiet place. After a few months the patient may be permitted to add a few words to the daily allowance but rigid control of the patient in this régime is necessary for a long time, even years in some cases.

*Dysphonia plicae ventricularis* calls for treatment according to the cause in the particular case.<sup>9</sup>



## CONCLUSIONS

1. The word benign in connection with disease of the larynx should always be used with caution and reservation; it should never be regarded as synonymous with innocent.

2. Practically all cases of cancer of the larynx start with a history (not necessarily a histology) of a benign lesion causing hoarseness.

3. The reverse should be the case; every case of hoarseness should be regarded as potentially serious in a child, potentially cancerous in an adult, until proven otherwise by every diagnostic means we possess. This is a good rule notwithstanding it will not work backwards. Absence of hoarseness does not exclude malignant disease.

4. Having excluded cancer the diagnosis and treatment of benign lesions causing hoarseness is quite simple and usually successful.

5. Most of the inflammatory diseases of the larynx are caused and perpetuated by vocal abuse; its elimination is the most difficult part of the treatment of benign lesions causing hoarseness. Infection, alcohol, tobacco, dust and the application of silver nitrate are the other causes.

6. The diagnosis of chronic laryngitis should never be made until every other diagnostic possibility has been excluded.

7. In every case of hoarseness the anterior commissure should be seen; if not visible in the mirror the direct laryngoscope should be used. Death often lurks under an overhanging epiglottis.

8. In removing benign growths of the vocal cords the edge of the cord should never be left concave if possible to avoid it; hence normal basic tissue should not be removed. It is better to leave a convex rather than a concave edge, even though this may involve leaving a vestige of growth for subsequent removal after sharper demarcation. Tension and approximation are more difficult tasks for the laryngeal musculature when the cord has a concave edge.

9. Contact ulcer and phonation with the ventricular bands are the most commonly overlooked causes of benign hoarseness.

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# AN ANATOMICAL AND CLINICAL STUDY OF CENTRAL LESIONS PRODUCING PARALYSIS OF THE LARYNX

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One has only to reflect upon the developments in laryngology during the past twenty years to realize that few, if any, basic or significant discoveries have been made in the neurological phenomena of the larynx. The problems which were discussed two decades ago in the didactic and clinical presentations of my senior year in the medical school, are yet unsolved and those studies in research which have been enthusiastically announced in our medical publications from time to time have failed, with but few exceptions, to live beyond that feeble period of existence which we term the preliminary report. The precise functions of the various elements of the laryngeal nerves are still under debate. The histological variations of the laryngeal nerves and of the muscles of the larynx are matters of scientific conjecture. The component parts of the central mechanism have not been identified to the complete satisfaction of scientists in this field. The physiological reactions of the nuclear cells still remain a mystery, and the character of the impulses which upon stimulation traverse the afferent and efferent fibers of the laryngeal nerves, is as obscure today as it was at the time when laryngologists first gave thought to a study of the activities of nervous tissue.

On the face of it then, and in spite of a few isolated examples of brilliant investigation, our record of achievements in research pertaining to laryngeal paralysis in the past twenty years is not an enviable one. There are innumerable studies concerning which we are much in the dark and many problems whose solutions will make desirable contributions to the science of laryngology. I emphasize this fact to show the difficulties one encounters in assembling knowledge of laryngeal paralysis which is not a matter of historical record. Actually, however, we have a comprehensive grasp of the clinical manifestations of laryngeal paralysis, but this has not been acquired within the past two decades. The pioneers of laryngology designated the etiological factors, described the symptomatology and established principles of treatment, which for the most part do not require revision or modification as the result of any modern discoveries of basic

significance in this field. All of which again emphasizes my point of view that any effort on my part to correlate and publish our present-day clinical knowledge of laryngeal paralysis would result merely in a repetition of medical history. The subject might lend itself admirably to embellishments and glorification but the product remains unaltered and the method open to censor, and properly, too, by those who hold priority rights to the original publications.

It is not my intention, therefore, to give you a didactic discourse on paralysis of the laryngeal nerves. I need not point out to you the reasons for laryngeal paralysis following operations upon the thyroid gland. I need not take your time to tell you that mitral stenosis with its consequent dilatation of the left auricle may cause paralysis of the left inferior laryngeal nerve, that mediastinal tumors, tuberculous fibrosis of the left lung and suppurative processes in the cervical region may produce a similar effect. It would be presumptuous on my part to describe to you the appearance of the larynx in the various forms of laryngeal paralysis. These are clinical facts exhaustively treated in our medical textbooks and well known to all of you. I do not believe that such a challenge was implied in the invitation which the president gave me when he entrusted to me the responsibility of presenting this subject.

There are, in any scientific discussion, many acceptable methods of presenting a subject but one which invariably attracts my interest is the plan of combining an anatomical study with the clinical manifestations of disease. I believe that we frequently lose the vital concepts of a disease entity by our failure to correlate what we know anatomically with what we observe clinically. Any medical problem backed by indisputable anatomical facts and supported by clinical example is, in my opinion, more clearly understood, more logically classified and better accredited by those who have given thought to it.

This method with its apparent advantage seemed particularly adaptable to a study of laryngeal paralysis. For obvious reasons I shall not attempt to apply it to lesions affecting the peripheral nerve trunks. As previously stated the peripheral pathways of the laryngeal nerves and the location and character of the morbid processes which affect them are so well recognized and so admirably presented in our textbooks and medical literature that further reiteration on these points would only serve to squander your time. However, in the matter of the intracranial distribution of the fibers of the laryngeal nerves there is much to be learned and many pertinent facts to be acquired by the plan of study above described. In this



connection I have attempted to outline in a general way the applied anatomy of the laryngeal nerves within the central nervous system and to designate the possible locations of lesions affecting the nerve fibers along their entire course from their cortical cells of origin to their sites of exit from the brain stem. In most instances I am privileged to illustrate my points by clinical records and by pathological sections obtained at autopsy. Needless to say a study of this character entails something more than a mere consideration of the laryngeal nerves. Central lesions are prone to involve neighboring structures and to produce neurological syndromes so that a recognition of associated paralysis is essential to a study of this kind. I will, therefore, direct your attention to the various neurological aspects of the lesions under discussion, keeping as it were the laryngeal nerves predominantly in the picture and giving consideration only to those nerve disturbances which with those of the laryngeals comprise a neurological complex.

#### ORIGIN, COURSE AND DISTRIBUTION OF CORTICOBULBAR FIBERS

Corticobulbar fibers carrying motor impulses to the lower motor neurons which supply the larynx have their cells of origin in the Betz cells or giant pyramidal cells in the lower part of the precentral gyrus of the cerebral cortex. The neuraxes of these cells pass as medullated fibers through the genu region of the internal capsule, enter the cerebral peduncle with other corticobulbar and corticospinal fibers in the middle three-fourths of that fiber mass, and below the peduncle interdigitate with the fibers of the pons. In the pons, part of the bundles swing dorsalward with other corticobulbar fibers as the pontine division of the aberrant pyramidal tract and continue caudalward in close approximation to the medial lemniscus system. Below the pons the corticobulbar fibers for the laryngeal group lie partly within the pyramids of the medulla and partly as aberrant pyramidal fibers intermingled with the ventral portion of the medial lemniscus systems. They terminate, *after a partial decussation*, in the middle portion of the nucleus ambiguus around the cells of origin for the superior laryngeal nerve and in the caudal part of the nucleus ambiguus around cells of origin of the recurrent nerve. Thus the impulses brought to the cells of origin of the recurrent nerve on the left side will arise from both the left and right precentral regions of the cerebral cortex and this will be true whether the fibers course in the aberrant pyramidal system or in the pyramids.

The above anatomical facts have an important clinical application in that lesions above the nucleus of origin of the laryngeal nerves (n. ambiguus) can produce a paralysis of the larynx only when they are large enough to affect the corticobulbar fibers on both sides in which event the paralysis is bilateral and of the spastic type. The decussation of the corticobulbar fibers renders supranuclear lesions incapable of producing a unilateral laryngeal paralysis. Lesions within the nucleus ambiguus or infranuclear in location will produce a flaccid type of paralysis, unilateral in character if one side alone is involved and bilateral only when both nuclei or both infranuclear pathways are affected.

It is frequently stated in our laryngological texts that central lesions usually produce a bilateral paralysis of the larynx. While this is probably true I am convinced that unilateral paralysis of central origin is not an uncommon condition. When both sides of the larynx are paralyzed phonation and respiration are seriously disturbed and the condition of the patient becomes so grave that the laryngologist is promptly consulted. In the case of a unilateral paralysis of the larynx, there is often but slight impairment of laryngeal function and because the associated neurological disturbances dominate the picture the slight alteration of voice which may be present is considered too trivial to merit investigation. A routine study of many neurological cases over a period of twenty years has brought to my attention many examples of unilateral laryngeal paralysis and only a few in which the central disturbance involved the laryngeal nerves on both sides.

### 1. *Description of Lesions.*

a. Large hemorrhages, tumors, abscesses or other lesions involving the cortical laryngeal areas on one side only will not produce paralysis of the laryngeal muscles because the corticobulbar path to the nucleus ambiguus (which gives rise to the motor fibers of the superior laryngeal and recurrens nerves along with other motor fibers) are both crossed and uncrossed. Destruction on both sides of the cortex at the appropriate regions would produce a bilateral laryngeal paralysis of the spastic type but such lesions are exceedingly rare. (It is to be remembered that supranuclear lesions produce a spastic paralysis while lesions within the nucleus or infranuclear in type produce a flaccid paralysis.)

a1. Lesions of the internal capsule (frequently hemorrhages) in the germ region on one side do not produce laryngeal paralysis for the reasons cited above.

b. Syphilitic lesions may often involve the brain in the region of the interpeduncular fossa. If such a lesion extends far enough lateralward to involve about four-fifths of the basis pedunculi a spastic paralysis of the muscles of the body and head (except possibly some eye muscles) and larynx will result. The emerging fibers of the oculomotor nerve will be involved, and a lower motor

paralysis of the muscles supplied by this nerve will be observed. Whether or not a spastic paralysis will be observed in the other eye muscles will depend upon the extent of the lesion.

c. A lesion (frequently syphilitic in character) may involve the paths in the basal part of the pons. Circumscribed lesions of this type which would result in a lower motor neuron paralysis of both lateral recti muscles (supplied by sixth nerve), and a spastic paralysis of both sides of the body. The larynx, however, would not be involved.

If the area of destruction is increased still further the aberrant pyramidal system may be involved on one side, in which case we will still observe that the laryngeal nerves continue to function in a normal manner. (If the medial lemniscus is involved proprioceptive disturbances of the trunk will appear.)

d. A frequent lesion occurs as a result of thrombosis of the posterior inferior cerebellar artery. A nystagmus would develop because of involvement of the vestibular area just medial to the inferior cerebellar peduncle. Interference with the descending root of the fifth nerve and its nucleus would lead to a loss of sensibility (particularly of pain and temperature, and possibly some tactile) from the lower part of the face. Interference with the lateral spinothalamic would produce a loss of pain and temperature (but not tactile) sensibility from the opposite side of the body. On account of the injury to the left inferior cerebellar peduncle, there would be difficulty in maintaining equilibrium, the patient showing a tendency to swing to the left side. Involvement of the nucleus ambiguus would lead to a lower motor neuron paralysis of the laryngeal muscles on one side of the larynx.

d. The spread of a meningitis about the pons would involve the nerve roots of both the tenth and the eleventh nerves and produce in addition to other paralyses, a paralysis of the laryngeal muscles. This, no doubt, is the common pathological change responsible for bilateral laryngeal paralysis in patients suffering from a syphilitic meningitis.

e. A lesion of the left lateral spinothalamic tract and the left nucleus ambiguus is occasionally the result of hemorrhage in the medulla oblongata. Such a lesion produces a lower motor neuron paralysis of muscles supplied by the left recurrent laryngeal nerve and the left vagus. The destruction of the lateral spinothalamic tract, resulting from this lesion, would also produce a loss of pain and temperature on the opposite or right side of the body. (Syndrome of Avellis.)

e. A lesion of the left nucleus ambiguus may produce a paralysis of the muscles of the larynx on the affected side (other striated muscles supplied from this nucleus through the ninth and tenth nerves may also be affected if the lesion is large enough). The nucleus ambiguus consists of a long band of motor neurons, the most cephalic of which supply fibers through the ninth nerve to the stylopharyngeus muscle. Next caudalward lie the cells supplying the vagus and the superior laryngeal nerves. The caudal end of the nucleus ambiguus supplies fibers through the internal ramus of ninth which joins tenth in the region of the jugular foramen and passes out with it to split off as the *vecunens* nerve. Within this caudal end of the nucleus ambiguus (as in other portions of the nuclear mass) the neurons are in groups. There is scientific reason for the opinion that this grouping is in accordance with adductor and abductor muscle function of the vocal cords and that the fibers arising from the nucleus retain this grouping in their course to the laryngeal muscles.

(Russell.) A lesion of this character may be produced by a tuberculoma. Occasionally the accessory nucleus of the cord is likewise affected by such a pathological change producing a paralysis of certain muscles of the neck. (Syndrome of Schmidt.)

e<sub>3</sub>. A lesion involving the right nucleus ambiguus and the right hypoglossal nucleus (which is occasionally syphilitic) sometimes produces a lower motor neuron paralysis of the muscles supplied by the right vagus, and (if large enough) by the right laryngeal nerve. Associated with the disturbed function of these nerves one may find paralyses of other striated muscles supplied by the vagus and of the stylopharyngeal muscle supplied by the glossopharyngeal nerve. There will be a lower motor neuron paralysis of the muscles of the tongue supplied by the right hypoglossal nerve, so that the tongue is drawn to the left side. (Syndrome of Jackson.)

e<sub>4</sub>. A lesion (produced, for example, by thrombosis or hemorrhage of the anterior spinal artery) may involve the right hypoglossal nerve, the right medial lemniscus, the right aberrant pyramidal tract and the right pyramid. In this type of lesion one will observe a disturbance in proprioceptive impulses and two-point discrimination from the right side of the body. The patient will present a spastic paralysis of the muscles of the left side of the body and a lower motor neuron paralysis of the right hypoglossal nerve. A paralysis of the laryngeal muscles will not be observed unless the lesion is so large that it involves all corticobulbar fibers crossing to the other side. The absence of laryngeal paralysis is due to the fact that the intact pyramid and intact aberrant pyramidal system on the left side carry fibers which cross to the right nucleus ambiguus as well as uncrossed fibers to the left nucleus ambiguus. It is apparent therefore that an injury on the one side will not produce a laryngeal paralysis. If the lesion spreads dorsolward beyond certain limits, there will be a loss of tactile sensibility on the side of the lesion. (Syndrome of Babinski-Nageotte.)

e<sub>5</sub>. A lesion involving both pyramids, both hypoglossal roots and both aberrant pyramidal fibers (supplying fibers to the nucleus ambiguus) produces a bilateral spastic paralysis of the body muscles, a bilateral paralysis of the laryngeal muscles and a lower motor paralysis of the muscles of the tongue. (e<sub>5</sub> should be compared with e.)



# LATE CHANGES IN THE MUCOSA OF THE FRONTAL SINUSES AND NOSE OF DOGS FOLLOWING IONIZATION WITH ZINC SULPHATE SOLUTION\*

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The value of ionization as a sound method of treatment of allergic conditions of the nose should be judged, not only by the amount of relief from local symptoms which is supposed to follow, but also by the possible deleterious effects which it might bring about in the tissues ionized. We have too often adopted unsound empirical methods solely because of our enthusiasm for something new or sensational, and have eagerly rushed in to apply the method of cure while it was still curing.

There are certain fundamental laws of tissue reactions which may not be ignored, and those governing the reaction of repair in connective tissue are the most inflexible. Our ever-present anxiety to prevent adhesions and synechiæ following our operative procedures, is evidence of our tacit respect for these laws. While we readily acknowledge the interference with function that is caused by fibrosis within the parenchymatous visceral organs, we do not appreciate the fact that adhesions, fibrosis, may be activated within the tissues of the nose as well as between the gross structures by undue irritation, and thus cause serious interference with the proper functioning of these tissues.

From our experiments of last year<sup>1</sup> certain conclusions were arrived at, namely, that definite destructive changes were brought about in the mucosa of the frontal sinuses of dogs by the ionization with zinc sulphate solution. These changes consisted of ballooning, fragmentation and complete destruction of the surface epithelium in most places, a marked edema and partial disintegration of the subepithelial tissues, and an extravasation of free red blood cells into these tissues from greatly dilated and ruptured capillaries.

As a result of these extensive changes, we felt that in the process of regeneration of the mucosa more extensive permanent changes might be brought about, and it was with the idea in mind of studying the possible progressive changes, that these experiments were pursued.

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## PROCEDURE

Healthy dogs, free of nasal infection, were selected and prepared by injecting intraperitoneally 6 to 10 c.c. of amytal solution, according to the size of the dogs. The electrode used was composed of zinc 85 percent; tin 10 percent; and cadmium 5 percent. The solution ionized contained zinc sulphate 8.92 grs.; stannic chlorid 1.42 grs.; and cadmium chlorid 0.45 grs. to the ounce.

The choanæ were blocked with a postnasal plug composed of a rubber finger cot filled with cotton, and held in place with an attached string passed through the unionized side of the nose. The electrode, thinned to prevent trauma and wrapped with long fibre cotton, was carefully introduced into the one side of the nose and this side filled with the solution so that every portion of the mucosa would be affected, as it is impossible to completely pack the nose with cotton without causing considerable trauma. The galvanic current, 2.5 milliamperes, was permitted to flow for twenty minutes, the positive electrode in the nose, the negative electrode completing the circuit at a shaved area on the back of the neck. This routine was followed in Dogs 1, 3 and 4, while in Dog 2, the wrapped electrode, kept saturated with the solution during the time of the ionization, was used without a postnasal plug. In Dogs 5 and 6, both frontal sinuses were filled with the solution and ionized at 2.5 milliamperes for twenty minutes.

The dogs were killed at definite intervals (*vide* chart) the heads placed in formalin, the nasal or sinus blocks cut after twenty-four hours, decalcified, stained with hematoxylin and eosin and serial sections cut.

## MICROSCOPIC FINDINGS

*Dog 1.*—Right side of nose ionized. Killed in eighteen hours.

*Epithelium:* Cells higher than those on unionized side, elongated on account of granular fluid in protoplasm, and vacuolization. Fragmentation of individual and large blocks of cells with round cells in large numbers infiltrating through to the surface from the subepithelial tissue. Cilia generally unrecognizable.

*Subepithelial Tissue:* Edema moderate to extreme: Round cell infiltration mild to marked. Entire tissue fragmented and separated from periosteum in various places.

*Glands:* Swollen or fragmented.

*Blood Capillaries:* Engorged generally, some ruptured, large numbers of free red blood cells in many places.

*Dog 2.*—Right side of nose ionized, wrapped electrode alone used. Mucosa grayish pink along tract of electrode. Killed in two weeks.

*Epithelium:* Definitely thickened and more concentrated than on unionized side, also higher; in other areas more goblet cells are seen; cilia present.

*Tunica Propria:* More mature diffuse connective tissue cells in large numbers throughout, but especially in septal areas, and more fibrous strands of connective tissue present. Blood capillaries dilated throughout.

*Dog 3.*—Right side of nose ionized. Killed in four weeks.

*Epithelium:* Higher than on unionized side; intact: usual number of goblet cells present. Cilia present.

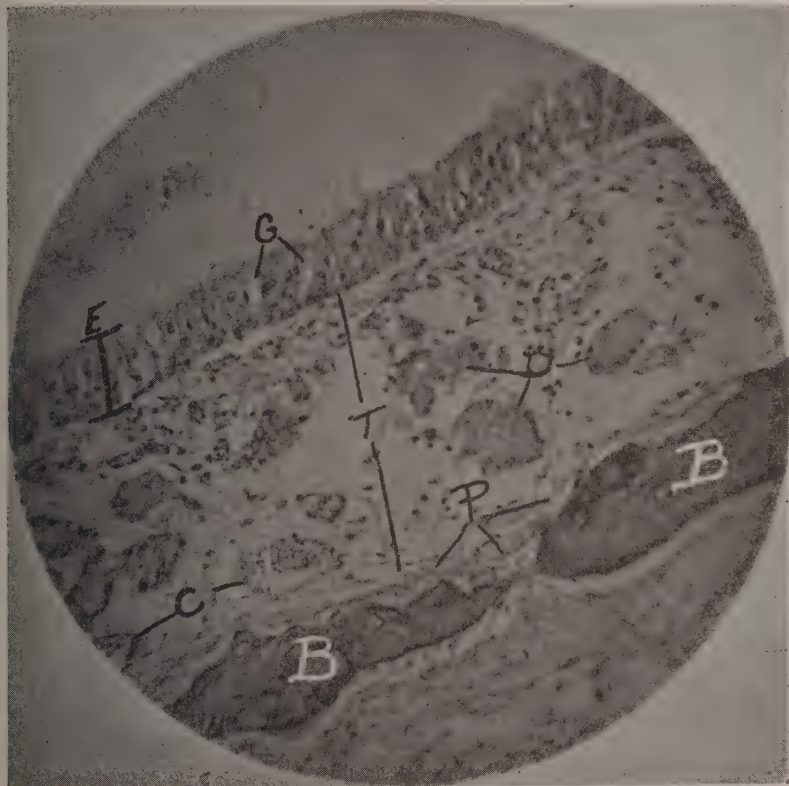


FIG. 1. Normal mucosa of turbinate of dog. E, epithelium—stratified columnar, ciliated; G, goblet cells; T, tunica propria—connective tissue stroma of normal density; D, glands; C, capillaries; B, bone; P, periosteum.

*Tunica Propria:* Thickened by broadening of the connective tissue strands of the stroma, especially adjacent to periosteum, and of the supporting tissue of the glands, and by moderate to marked connective tissue cell infiltration. *Vessel walls* thickened.

*Dog 4.*—Left side of nose ionized. Killed in seven weeks.

*Epithelium:* No appreciable change from normal on either side.

*Tunica Propria*: Noticeable thickening with marked connective tissue cell infiltration in most places with fibrosis especially adjacent to periosteum. Nerve trunks midway between epithelium and bone. Smaller trunks more superficial in glandular layer. Fibrils everywhere even in epithelium in places and especially along periphery of walls of blood vessels. No degeneration of axones or myelin sheaths discernible. (Ranson's pyridin silver nerve stain.)

*Glands*: Cells more swollen to point of obliteration of lumen in most cases, especially in areas of most intense cellular infiltration.

*Blood Capillaries*: Some thickening of walls.



FIG. 2. Mucosa of septum and adjacent turbinate—eighteen hours after ionization. X, exudate—serofibrinous; E, epithelium, swollen and partially disintegrated; Tp, tunica propria, markedly edematous: infiltrated with young connective tissue cells in subepithelial zone; C, blood capillaries—greatly dilated, some ruptured; G, glands—edematous; S, septum; T, turbinate; B, bone.

*Dog 5*.—Frontal sinuses prepared as in previous experiments. Positive electrode in each. Killed in seven weeks.

*Epithelium*: Normal and higher than normal, ciliated, large numbers of goblet cells except over windows where it is a non-ciliated, low cuboidal type.

*Tunica Propria*: Marked fibrosis in most places with and without thickening; less fibrosis in other areas; frequent areas of round cell infiltration.



*Blood Capillaries:* Not changed.

*Periosteum:* Moderate to marked thickening.

*Dog 6.*—Each frontal sinus ionized with positive electrodes. Killed in twelve weeks.

Left sinus—opaque; right sinus—clear.

*Left:* Bony trabeculae throughout except for small rudimentary sinus cavity in center loosely lined with a delicate membrane containing few thin- and thick-



FIG. 3. High power magnification—mucosa of septum and exudate, shown in Fig. 1. X, exudate closely attached to epithelium; E, epithelium, swollen and partially disintegrated; Tp, tunica propria, markedly edematous; infiltrated with young connective tissue cells in subepithelial zone; C, blood capillaries—greatly dilated, partially ruptured; G, glands—edematous.

walled capillaries, and covered with single layer of pale staining low cuboidal epithelium. Other slides showed an ill-defined cavity filled with fibrinous tissue, more concentrated at margins of irregular bony trabeculae. No epithelium. Many thin-walled capillaries of various sizes.

*Right: Epithelium:* High columnar ciliated—large numbers of goblet cells. Low cuboidal—almost squamous—in places interspersed with connective tissue cells—non-ciliated—over area of bone regeneration of window—similar type at other areas.

*Tunica Propria:* Thickened—edematous in places—and infiltrated with large numbers of round and connective tissue cells, fibrosed and contracted in other places. Large numbers of new formed blood capillaries and blood spaces of all sizes.

#### MICROSCOPIC FINDINGS AFTER IONIZATION

	1	2	3	4	5	6
	18 hrs.	2 wks.	4 wks.	7 wks.	Sinus 7 wks.	Sinus 12 wks.
Epithelium:						
Fragmentation .....	**	..	..	..	..	..
Thickening .....	***	*	*	*	*	*
Metaplasia .....	..	..	..	..	P	P
Cilia .....	..	P	P	P	P	P
Tunica propria:						
Edema .....	***	..	..	..	..	**
Fragmentation .....	**	..	..	..	..	..
Thickening .....	***	*	*	**	**	***
Young connective tissue cells .....	*	***	**	***	**	**
Fibrosis .....	..	*	**	***	**	***
Capillaries .....	D-R	D	N	D	N	D-I
Glands .....	D	D	C	D-C	..	..
Periosteal thickening.	..	..	*	**	**	***
Hyperostosis .....	..	..	..	..	..	***

\* Mild. \*\* Moderate. \*\*\* Extreme. C—Compressed. D—Dilated. I—Increased. N—Normal. P—Present. R—Ruptured.

From the foregoing chart the progressive nature of the fibrosis of the tunica propria of the mucosa of the nose, as well as of that of the frontal sinuses, is plainly evident. We may readily follow the steps of connective tissue reconstruction, from the time of irritation of the tunica propria by the ionization, which caused the laying down of fibrin and the entrance of the very young connective tissue cell which became the fibroblast and then the mature connective tissue cell (Figs. 3 to 7), and finally, we witness the agglomeration of these cells to form fibrous tissue, which is a definite and permanent structure (Fig. 9). This progression was gradual, but unmistakable, starting in the dog killed a few hours after ionization through five dogs killed at intervals, to become fully established in the dog killed twelve weeks after ionization.

The frontal sinuses were again used in these experiments in order to complete our observations of last year, inasmuch as the destructive changes noted in the mucosa immediately after ionization prognosticated reparative changes of a character deleterious to the proper functioning of the mucosa. The reaction of the connective

tissue of the tunica propria of the sinuses was identical in intensity to that of the nasal mucosa (Figs. 6 and 7), and in direct proportion to the amount of ionization and to the length of time that the dog was permitted to live. The most intense reaction to irritation occurred in the left sinus of the dog killed in twelve weeks, in which practically the entire area of the sinus cavity was filled with trabeculae



FIG. 4. Septum and turbinate—two weeks after ionization. S, septum; U, unionized side; epithelial and tunica propria normal; I, ionized side: epithelium normal and cuboidal. Tunica propria—thickened, early fibrosis generalized. Marked round cell and young connective tissue cell infiltration in subepithelial zones; T, turbinate—epithelium—slightly thickened. Tunica propria similar to that of septum; N, nerve; B, bone.

of new formed bone, leaving no true sinus cavity, but a space partially filled with fibrin undergoing fibrosis and lined in places with an indifferent type of epithelium. The tissue underneath this epithelium and between the bony trabeculae consisted of a delicate granulation tissue (Fig. 8). In the tunica propria of the right sinus of the same dog, there appeared to be an increase in the number of the



blood capillaries, which seemed to take place by budding and branching from the original capillaries, as a reaction to irritation. This modification of the capillaries in the nasal mucosa was not noted, the only change being a slight thickening of the vessel walls. The fibrosis was most marked immediately underneath the epithelium, and in many places formed a solid band between the epithelium and the bone (Figs. 8 and 9).

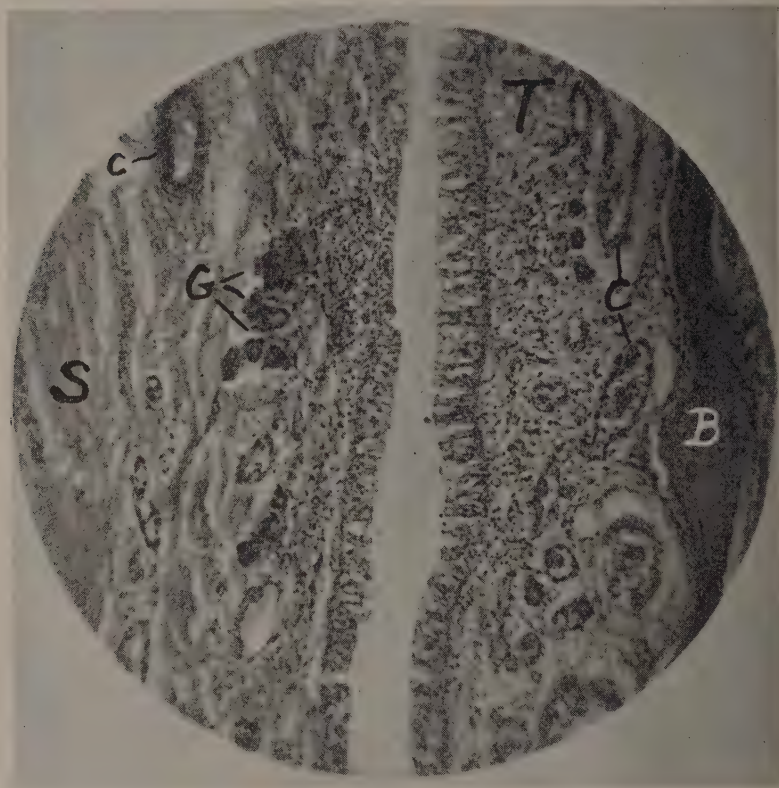


FIG. 5. High power magnification, Fig. 4, ionized side of septum and turbinate. S, septum. Epithelium—normal in lower  $\frac{3}{4}$ —changed to cuboidal in upper  $\frac{1}{4}$ . Tunica propria—marked round and young connective tissue cell infiltration in subepithelial zones—thickening of connective tissue stroma in deeper portions; G, gland—dilated; T, turbinate—epithelium thickened—large numbers of goblet cells. Tunica propria—marked cellular infiltration: more advanced fibrosis; C, blood capillaries; B, bone.

The epithelial reaction in the nose consisted principally in a granular degeneration of the nucleus and protoplasm of the individual cells and a disintegration and sloughing of the cells singly and in large clumps up to eighteen hours. This epithelium appeared to



have regenerated and returned to normal after two weeks. The surface was covered with a thick fibrinous exudate in which the sloughed epithelial cells were incorporated (Figs. 2 and 3). The epithelium of the frontal sinuses, which was presumed to be more or less completely destroyed by the ionization, had regenerated in its normal state, namely, a high columnar ciliated type containing many goblet

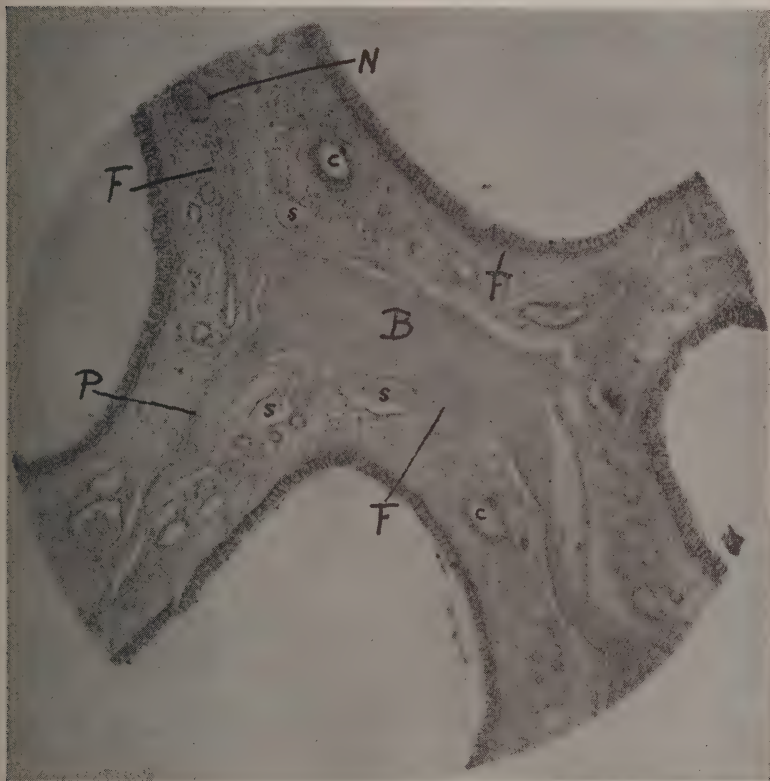


FIG. 6. Turbinate—seven weeks after ionization. Epithelium: normal. Tunica propria—thickened and markedly infiltrated with older connective tissue cells; more advanced fibrosis immediately underneath the epithelium and adjacent to the thickened periosteum; F, fibrosis; P, periosteum; C, blood capillaries—showing thickening of walls; S, blood spaces; B, bone; N, nerve.

cells, except over the areas of the healed operative windows and several other smaller areas where it was a non-ciliated low cuboidal, almost squamous, type interspersed in places with connective tissue cells (Figs. 7 and 9). This picture suggests that after the epithelium of a sinus is destroyed it does not regenerate entirely normally, even

though normal epithelium is present. It also points to the fact that though a sinus in which the mucosa has been destroyed may be lined with a partly normal epithelium, the subepithelial tissue is not normal as a result of the excessive fibrosis occurring in the reaction of repair (Figs. 7, 8 and 9).

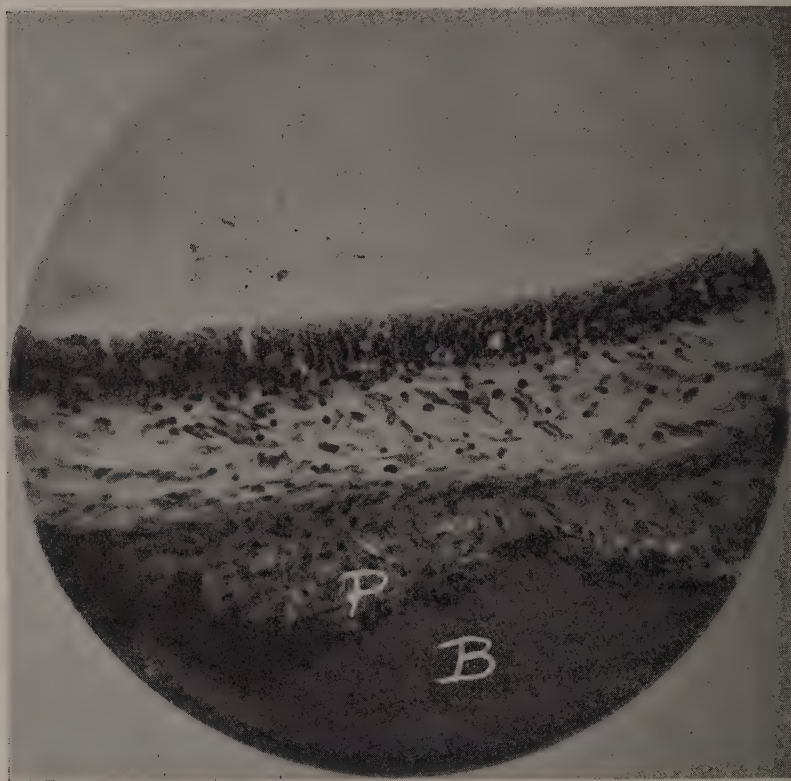


FIG. 7. Mucosa of frontal sinus—seven weeks after ionization. Epithelium—thickened and infiltrated with young connective tissue cells. Goblet cells absent. Tunica propria—greatly thickened and markedly infiltrated with older connective tissue cells, especially adjacent to periosteum; P, periosteum—greatly thickened; B, bone.

Our desire to ascertain whether or not ionization might affect the nerves of the nasal mucosa, prompted us to make special nerve stains (Ranson's pyridin silver) of a series of sections from the tissue of the seven weeks dog. The larger nerve trunks were located midway between the epithelium and the bone, the smaller trunks more superficially in the glandular layer and immediately beneath the

epithelium, and the fibrils arranged in a more or less irregular network throughout the entire mucosa, and along the outer coat of the blood vessels and capillaries. Occasional fibrils could be traced into the epithelial layer. No degeneration, either of the axones or the myelin sheaths, could be seen.



FIG. 8. Mucosa of right frontal sinus—twelve weeks after ionization. Epithelium—low cuboidal non-ciliated instead of the normal ciliated columnar. Tunica propria—markedly fibrosed immediately underneath epithelium and edematous with many new formed blood capillaries in deeper portions; P, periosteum—thickened; B, bone; F, fibrous tissue; C, blood capillaries.

### SUMMARY

We have seen the effects upon the mucosa of the noses and frontal sinuses of dogs following ionization; the immediate widespread destructive changes, and the readjustment of these delicate tissues, characterized by a progressive accumulation of connective tissue in



the tunica propria, as evidenced in dogs killed eighteen hours, two weeks, four weeks, seven weeks and twelve weeks, after ionization. The changes brought about in the frontal sinus mucosa and bone are valuable as a further corroboration of the reaction of such tissues to an extreme irritation.

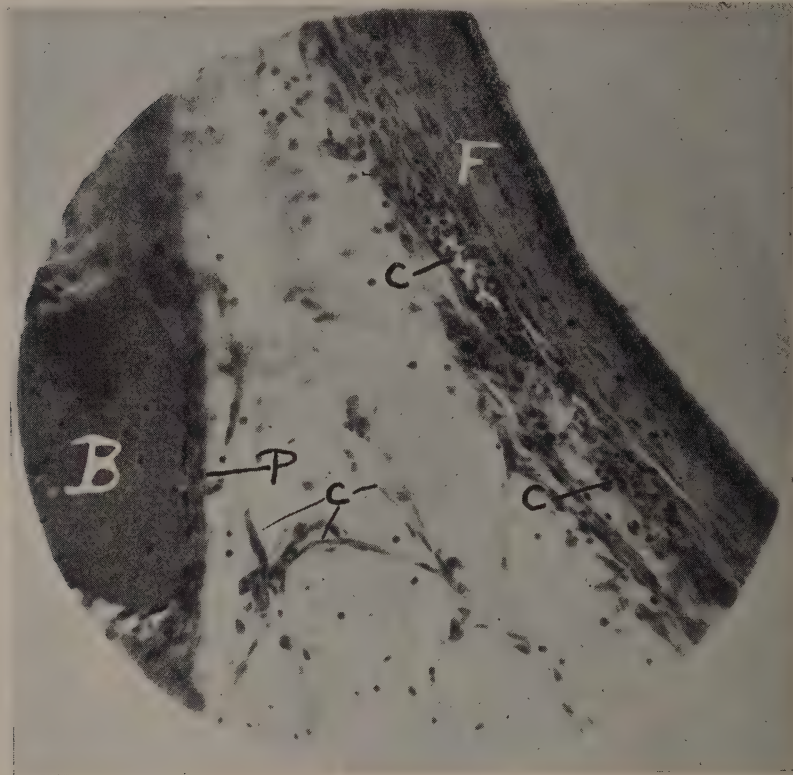


FIG. 9. Septum between frontal sinuses twelve weeks after ionization. R, right sinus; L, left sinus. E, epithelium—normal, bound closely to bone by a dense fibrous tunica propria; B, bone—trabecula extending into cavity of left sinus which is filled with young fibrous granulation tissue. Other slides show entire sinus cavity traversed by similar bony trabeculae; F, fibrous granulation tissue; C, blood capillaries; S, bone space partly filled with fibrous tissue; T, trabeculae.

From a clinical standpoint, however, we are concerned chiefly with the changes in the mucosa of the nose. Should we consider generalized fibrosis of the nasal mucosa as an optimum state for the proper functioning of that mucosa? Increased and excessive fibrosis means depletion of the blood supply to these structures and further fibrosis, it means compression atrophy of the glands and depression of secretions, and compression of nerves with the imminent



probability of giving rise to nasal pains, hyperesthesias, neuritis, and headaches of a more or less intractable nature, even though there was apparently no damage to the nerve structures themselves.

We would not expect all of these changes to take place within a few months in the noses of human beings which have been ionized, as the individual reaction is too variable as to time, but we have ample reason to fear that within a comparatively short time a condition would be brought about which might be aptly termed a premature nasopause.

### CONCLUSIONS

1. Ionization of the noses of dogs with zinc sulphate solution caused a fibrosis of the subepithelial tissues which was seen to be progressively more marked and well established in dogs killed at intervals of eighteen hours to seven weeks, following ionization.

2. Ionization of the frontal sinuses caused a similar fibrosis in dogs killed at seven and twelve weeks, and marked hyperplastic bone changes occurred in the dog killed at twelve weeks.

3. The epithelial changes in the nose consisted of a primary destruction and later regeneration practically to normal; and in the frontal sinuses of an eventual transition to a cuboidal type over the areas of greatest destruction.

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### COMBINED DISCUSSION ON "IONIZATION FOR NASAL ALLERGY" (page 109) AND "LATE CHANGES IN THE MUCOSA OF THE FRONTAL SINUSES AND NOSES OF DOGS FOLLOWING IONIZATION WITH ZINC SULPHATE SOLUTION"

ARTHUR M. ALDEN, M.D., St. Louis, Mo.: Doctor Hurd has given you a masterful presentation of the physics involved in ionization. His knowledge of this phase of the subject is so much more profound than is mine that I have not the courage even to discuss it.

Most of us started our work using the technic described by Warwick. Certain modifications are apparently of value. Quite independently I have also decided that the flexible metal strip protected by a wet pack is a much better negative electrode.

Early in our work with children we were forced to the determination of some basis upon which dosage might be computed. Our scheme at present is to give

patients weighing less than one hundred pounds a M.A. per pound plus 10 percent. Patients weighing more than 150 pounds, a M.A. minute per pound less 10 percent. Between these two weight limits they received a M.A. minute per pound. I have given as much as 170 M.A. in each side of the nose without additional reaction.

All of our children are done under avertin given upon a basis of 90 m.g. plus, when it is necessary, a very small amount of chloroform. This is quite a satisfactory anesthesia.

I have had no experience with the liquid contact in the nose but if the technical difficulties incident to its use can be removed, it would seem to be a distinct advance in that it would prevent the ciliary trauma which follows even the most careful packing.

The end-results Doctor Hurd has reported are about the same as the rest of us have observed, and I am quite sure that he will tell you that he has not as yet had to apologize to a patient for symptoms which might in any way be referable to the mucosal trauma incident to his ionization.

RALPH A. FENTON, M.D., Portland, Ore.: Doctor McMahon has in this contribution completed his accurate and scientific histopathologic experiments regarding the mucosal and submucosal results from so-called "ionization" with zinc sulphate solution.

Bombardment of the nasal mucosa by the galvanic current using a zinc electrolyte has been shown not to cause permanent change or damage thus far to the ciliated epithelium. In this respect, its effects resemble those of therapeutic roentgen ray irradiation.

In our experimental studies on such irradiated membranes, we found at first a release of immune substances through the killing of round cells in the submucosal stroma, with consequent mobilization of phagocytic connective tissue cells. Later changes exactly paralleled those shown here today; namely, a vast increase, beyond the normal amount, in fibrous connective tissue.

Now, if it be desirable to shrink the mucous membranes of the turbinates, tying them down to the underlying bones by fibrotic bands which will grow stronger as years go on, well and good. We have, however, all seen the late effects of excessive fibrosis due to other sorts of cauterization. Let us think well whether it is wise to risk destruction of the vascular defensive apparatus and of its sympathetic nerve endings, without in any way changing the individual's specific allergic sensitiveness as demonstrated by skin reactions or eosinophilia.

Doctor McMahon's attitude of scientific detachment remains in marked contrast to the indiscriminating enthusiasm for this potentially dangerous procedure manifested by some of its proponents. We pointed out this danger respecting roentgen irradiation in careless hands, two years ago. Already commercial organizations are utilizing communications from our members to bolster up their selling campaigns. We must not forget that these unnecessarily complex instruments for applying a simple current are readily obtainable by the irregular practitioner and the conscienceless charlatan.

Let me express the hope that Doctor McMahon may secure some human tissue within a year or two following this procedure, for study and dispassionate analysis of existing allergic reactions at that time, as well as alterations in the vascular stroma by which the epithelium is nourished. He is entitled to our heartiest thanks.

JOHN J. SHEA, M.D., Memphis, Tenn.: My first experience with ionization was as a house doctor in the experiments carried out by Doctor LeFevre in the treatment of syphilis by ionization with mercury in Bellevue in 1912. When, later on, it became popular for ionization of ears, I bought myself one of the new galvanic outfits and tried ionization of ears, with the experience that you could dry up a certain number of ears, but, if you took those same cases and did a radical mastoid on them, deep in you found that the pathology was still present and you had merely made a good show window and had not cleaned up the factory.

So, when Doctor Warwick started his presentation on ionization, we were interested. We who live in the Southwest have a long season of pollen. In the country below us it lasts as long as nine months with certain pollen, and patients with vasomotor rhinitis make up a large part of our unsatisfied clientele, so in order to test out the efficacy of Doctor Warwick's treatment, one of our fellow townsmen, Doctor Blassingame, went down to learn the technic from Warwick himself, and three or four of us chipped in and got machines to try it out to see what we could do.

Now, I will say that we have not yet compiled our local results in Memphis except that in the hands of the men who are using the method, they have felt that clinically they have satisfied the patients who were treated during the hay fever season time.

The patients who get the best results are those who are treated after they start their hay fever. For one day they have intense nasal obstruction that can be controlled by a moderate amount of sedative, then in three or four days the nose begins to open up and the mucous membrane changes from the pale vasomotor color to what is a normal pink to the eye, and for the rest of the pollen season the patient is comfortable, if a good ionization has been done.

I have only had the opportunity of taking tissue from one vasomotor patient. I think there is a difference in the reaction on a human vasomotor membrane and on a dog's membrane that hasn't had years of this defensive process going on. In defense of ionization, I will merely state that clinically it is popular in the part of the country where there is a long season of pollen, and these patients are clinically satisfied, and instead of their going to the allergist and out of the hands of the otolaryngologist, something can be done to help them.

WILLIAM B. CHAMBERLIN, M.D., Cleveland, Ohio: This method, if successful, will certainly offer a boon to us with those patients who come during the hay fever season. Granted they might be benefited or in some cases even cured by inoculation, time and experience have well proven the fact that inoculations given after the beginning of the hay fever attack are of no value.

Now, there was just one point which Doctor Hurd mentioned and on which I, at least, would like to have further enlightenment from the men who have had clinical experience in this procedure. I am free to confess that I have not.

Doctor Hurd mentioned the question of anosmia. That to me is a very significant remark and I should like to know the experience of Doctor Alden and Doctor McMahon, and others who have had experience with a number of clinical cases, as to whether or not they have had anosmia, and, if they have had anosmia, how long this anosmia has continued or if it has been permanent.

Certainly, if we are going to get a permanent anosmia, it is a very vital contra-indication to the use of the method.

JOSEPH C. BECK, M.D., Chicago, Ill.: We must all agree that there is a great difference between animal experiment on a dog and a human being, yet the changes that were shown by Doctor McMahon as to the vascularization will not vary whether in a pathologic preparation or a normal one or in an animal; in other words, we know what follows in a persistent vascularization, as was shown in these animals and that we should expect certain changes around the glandular elements and nutritional elements in the tissues, but it is too early, as far as we have gone, from the time Doctor Warwick presented his material, to this day, to see what is going to happen.

I want to ask Doctor McMahon whether he made a control of the other sinus, not by ionization but by simply a surgical procedure such as irritation, packing, or whatever it might be, and see what change occurred, so that it would be really not comparing a normal membrane to one that was ionized.

I want to state here something that I have done regarding the suggestion by Doctor Richards and that is, using the fluid and blocking the postnasal space by occlusion of the postnasal choana. Through a tube you can do it much more easily than with tampons, and with a child, using postnasal tampons is about as bad as packing or an operation.

CHARLES D. BLASSINGAME, M.D., Memphis, Tenn.: Individuals with hay fever are unfortunate individuals, and if they are not 100 percent improved after the treatment, if they are better at all, then our treatment will justify itself—the end will justify the means.

As Doctor Shea indicated, before I ionized a patient, after I had bought my machine, I went over to Fort Worth, with a view to talking with Doctor Warwick about his technic. Since then I have ionized about a dozen cases and my experience has been, I should say, satisfactory.

In regard to the anosmia, one patient that I have ionized says she has lost her sense of smell, but she says that that condition is much preferable to the hay fever that she formerly had.

It may be that in packing the cases, if we would not pack in the roof of the olfactory recess, we might avoid this complication.

SAMUEL R. SKILLERN, JR., M.D., Philadelphia, Pa.: I just want to ask Doctor McMahon whether or not in packing these frontal sinuses he packed actually into the frontal sinus or whether he packed simply through the nose. We have found in Philadelphia what we think is a rather new condition, *i.e.*, an obliterative frontal sinusitis from a proliferative osteitis.

If in twelve weeks' time ionization will start proliferation of new bone growth in the frontal sinuses by ionizing through the nasal chambers, are we going to get complete filling of the frontal sinuses with the new bone growth? If we do, we will get walled-off foci of infection, because, as those sinuses narrow down, if infection occurs, it stimulates further bone growth, and you get interosseous infections which are pretty difficult things to diagnose, and also they require the most radical surgery to clean them up; so, I am wondering if just the removal of frontal and ethmoidal polyps starts up this proliferative osteitis, whether this ionization is going to do the same thing. If it does, we will have to stick to the packing because the free fluid will certainly get into the sinuses and start a proliferative osteitis.

THOMAS C. GALLOWAY, M.D., Evanston, Ill.: This subject is apparently under consideration by the American Medical Association Laboratories and we



had a little discussion by Doctor Leech, the physicist, who says, in the first place, that ionization is not essentially different, probably, from the introduction of a more concentrated solution. He said that the use for ionization of 2 percent zinc sulphate solution is perhaps the same as having in contact with the mucosa a 20 percent solution, and it is possible, he thinks, we may reduce this to a much more simple procedure.

He says also that he can see no physical reason why a combination of metals for electrodes is of any additional advantage over zinc alone.

I wish some of the speakers who have considered the theoretical aspects of this case could tell us exactly how ionization works. Is it the destruction of the absorption surface for allergens, is the introduction of the reflex arc, or is it a diminution effect on the blood vessels themselves? I wish we could have some scientific work on that subject.

LEE M. HURD, M.D., New York, N. Y. (closing): Doctor Titus, of New York, has over a long period of time treated hay fever by the ultraviolet ray and he has 97 percent of relief.

I can't remember the name, but I know a man packing the nose and raising the temperature, who has relieved his patients.

The flooding game is pretty and nice, but we are getting anosmia! One woman has a paranasal now.

And another point: If we are knocking out the olfactory nerve, we are knocking out the sensory nerves of the nose at the same time and that is the reason they don't have symptoms.

As far as this flooding game goes, it is much easier to do it just the opposite way. I have a curved glass nasal tip and a piece of rubber tube over it put in the nose. The patient holds it in the nostril and you can see the electrolyte level, and when the nose is full on that side the solution will run out the other side, and you are ready.

What we are trying now is packing olfactory clefts with vaseline gauze to see if we can occlude them from the ionization.

About this combination of metals, I think we have settled that. There is no tin. Professor Muller, of New York University, examined the first lot and found no tin.

We have used commercial electrolyte on one side and zinc sulphate on the other side, and we see no difference on the two sides afterwards.

BERNARD J. McMAHON, M.D., St. Louis, Mo. (closing): Doctor Shea's slides brought out the point that the changes in the mucosa are really nothing more than the result of trauma, whether it is an operative procedure, zinc sulphate, or phenol, or whatever other type of irritant you wish to use. The reaction of the tissues to excessive handling is a fibrosis; it is in the connective tissue reaction we are interested, and his slide showed a definite, marked, connective tissue reaction, fibrosis, and that follows any irritation.

The fluid solutions were used for the reasons which I outlined, and the frontal sinus mucosae were not changed. Of course, we sectioned all the way through and took the frontals in with the nose and it showed no change in the mucosa. I don't see why we should be afraid of going up into the sinuses as long as we have no displacement of air in the sinuses as proven by Doctor Proetz's work.

I cannot answer the question about anosmia. I have had no clinical experience with ionization. I don't feel justified in following it out yet, though I can

easily see why it would take place because it is a known fact the ions of the heavy metals do destroy the nerve endings; whether that is permanent or temporary, we don't know, as we haven't followed the cases for a long enough time.

I think it is possible that ionization may have a direct and valuable use because some solution may be worked out which will replace the salts lacking in the tissues of the nose or change the reaction, not forgetting that allergic rhinitis is only a local manifestation of a systemic dyscrasia.

As to Doctor Skillern's question, I evidently did not make myself clear. I didn't pack the frontal sinuses. A window was removed and was filled with solution and the electrode was introduced into the solution. The walls were not touched. I don't want to convey the impression that the hyperostosis was the result of ionization. It simply followed ionization in one case, showing that an extreme irritation does have an effect on the deeper tissues as well as on the bone.

## THE CARE OF CLEFT PALATE PATIENTS\*

By HUGH G. BEATTY, M.D.  
Columbus, Ohio

Experience in treating patients suffering from cleft palate makes it more and more evident that certain other matters are almost if not quite as important as surgery—important both in preserving the health of the child until operation and also in securing satisfactory functional results through surgery and through observation afterward. These matters are just as essential but not as often emphasized as some particular surgical technique.

No attempt will be made to evaluate the various methods of operation employed by different men except to illustrate some particular point. Such an attempt would be very presumptuous inasmuch as there have been and are so many men whose opinions and work along this line have been and are respected.

The opinions and suggestions to be offered in this discussion of the subject are based on the writer's own experiences in the treatment of three hundred and eighteen patients. Various types and extents of deformity require one or more operations; failures require subsequent correction. The number of operations in the series has been approximately three hundred and ninety-three.

Many of the patients have had a complicating cleft lip, but the correction of this deformity has not been included in the number of operations listed above. Including harelip cases, the total number of patients would be three hundred and forty-three and the operations would total six hundred and two.

In the chart, on page 166, the cases are classified with reference to the type and extent of deformity. While the total number is not as great as cited by many others, the relative occurrence of each type in this list is representative of the frequency of its presence in practice.

Many men have for years described in detail their technique so that anyone doing palate surgery has a large variety of methods from which to choose. The writer has taken from the various methods parts that seem to be the best adapted to his ability and that give at his hands the best results. As all surgeons do, he has made some modifications, but no individual type of operation is to be here described.

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\* Candidate's thesis.

## 132 Cases

## I. Complete Cleft of Alveolar Process, Hard and Soft Palate:

Unilateral—	Right	40.....	26.32%
	Left	70.....	46.05%
Bilateral —		42.....	27.63%

## 23 Cases

## II. Cleft of Alveolar Process Only:

Unilateral—	Right	4.....	17.39%
	Left	18.....	78.26%
Bilateral —		1.....	4.35%

## 93 Cases

## III. Cleft of Hard and Soft Palate Only:

Unilateral—	Right	0.....	0
	Left	5.....	5.38%
Bilateral —		88.....	94.62%

## IV. Cleft of Hard and Soft Palate:

(Complicated by incomplete cleft of one side of hard palate)

34.....	10.7 %
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## V. Cleft of Soft Palate Only:

16.....	5.03%
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Total .....318.....

I—47.8%. II—7.23%. III—29.24%. IV—10.7%. V—5.03%.

As Goethe said: "If I could give an account of all that I owe to great predecessors and contemporaries, there would be but small balance in my favor."

## HISTORIOGRAPHY

No attempt will be made to give a detailed historical review of the work since that has been done in such an exhaustive manner by Dorrance<sup>1</sup> in his recent work, "The Operative Story of Cleft Palate."

According to Dorrance, "Rolando Capelluti of the school of Serlane (twelfth century) has been mentioned by Velpeau and Tirifahy as having observed fissure of the palate. No treatment was suggested." The first mention by Dorrance<sup>2</sup> of an operation having been performed is Robert's report in 1766 that le Mormier, of Rouen, had successfully closed a complete cleft of the palate. An abstract of the description of the operation states that "M. le Mormier, a skillful dentist, succeeded in reuniting the borders of the cleft by first inserting several points of suture in order to keep them approximated and afterward abraded them with 'the actual cautery.' An inflammation supervened, terminated in suppuration, and was



followed by reunion of the lips of the artificial wound. The child was perfectly cured."

Dorrance<sup>3</sup> also states that Eustache in 1779 first mentioned the difficulties of deglutition and speech in congenital absence of the velum. Four years later (1783), Eustache described his operation for suturing the velum, split for the removal of nasal polyps (after the method of Manne), and added that this method could be applied in cases of congenital split of the velum. This then seems, according to Dorrance, to have been the first description of a technic for closure of the cleft palate—in 1783.

From this time, the literature carries various descriptions of technic of operations for this deformity—and still does. The old methods of securing union by cautery, inflammation, and suppuration have long been discarded; indeed many of the procedures advised today by various operators are not generally accepted by their more experienced colleagues.

The earlier writings are descriptions of various methods of "curing" harelip. This more noticeable deformity apparently was the first part of the deformity of harelip and cleft palate to interest physicians in making attempts at closure.

#### BIBLIOGRAPHY

1. Dorrance, George Morris: *The Operative Story of Cleft Palate*. Saunders, Philadelphia, 1933, p. 441.
2. *Ibid.*
3. *Ibid.*

#### PREPARATION OF PATIENTS

The surgeon selected to do the operations should see the patient soon after birth to outline his surgical procedure and, together with a competent, alert, well-trained pediatrician, direct the care of the infant up to and through the various operations necessary to correct the deformity.

We must remember that these patients are never an emergency, surgically. Therefore, we must be as certain as possible that they are in every way fit for operation—more so than in general surgery—as the operation, when correctly and carefully done, is necessarily longer than any other done on infants. We should employ all known methods of arriving at this conclusion. The pediatric advice together with the reports of the various laboratory x-ray and blood examinations, are very important.

None of these patients die before operation as a result of the deformity. If they die, it is because of faulty or incorrect feeding methods or an improper formula or some intercurrent disease.

The proper amount of the proper formula for the particular patient must be established before operation. A breast-fed baby must be taught to take food from some form of feeder before operation. An infant improperly fed, dehydrated, and with an almost necessarily upset gastro-intestinal tract is one of the poorest risks for operation.

The baby with a "thymus shadow" or any tendency toward visible lymphoid tissue hyperplasia (pharynx, etc.) in excess of normal causes a greater anxiety over the possible post-operative reaction than the ordinary child. These patients are given an unusually detailed study. For some time before operation, they receive iodine medication (Lugol's, etc.) or proportionate doses of thyroid extract; those with an enlarged thymus shadow are given several x-ray treatments, with the x-ray examination repeated before operation.

We have seen a number of patients of this type under the care of different pediatricians and their opinions differ on the subject. Fortunately we have observed severe reactions more times than death. Whether or not we believe in status lymphaticus as an entity, we do believe there is some secondary reaction in the glandular system in these patients. The endocrinologist interested in thyroid and thymus dysfunction tells us it is a lack of iodine balance and the failure of the thyroid to exert its government or direction of the body functions.

To the operator who has seen a baby expire in from five to twelve hours after operation in spite of all that can be done, this anxiety is real; especially so after he has gone through a careful pre-operative routine of examination and pediatric preparation. Many of these babies look well and seem in good condition, but often after a slight operation, temperature, pulse and respiration seem to be running wild without a governing force, and terminating in either death or several days of severe reaction. Further knowledge of the cause of this severe reaction is greatly to be desired.

The following is a history and post-mortem findings in such a case. Following closure of a cleft arch, the patient had been in the hospital for seventeen weeks under observation and pediatric care and was pronounced in as good condition as possible and satisfactory for operation. Although we knew the child was not normal in some ways, no positive contra-indication to operation was given, and the

family wanted the operation carried out. The child died eight and one-half hours after operation.

*Case.*—M. B., age nine months.

*Diagnosis.*—Right complete cleft of lip, alveolar process and hard and soft palate.

*History.*—Thirteenth child of mother, forty-seven years of age. First operation for repair of cleft of alveolar process at age of four and one-half months. Post-operative reaction following this operation was more severe than in the ordinary child. Respirations were irregular for the first two hours; temperature elevated from 102.8 (first hour) to 104 (fifth hour), but gradually subsided to 101.2 within twenty-four hours. At present (nine months) child does not sit up or hold up head, which is large. Weight: Fourteen pounds.

*X-ray of Thymus.*—"No enlargement of thymus gland."

*Laboratory Reports.*—Nose and throat cultures: Negative. Urinalysis: Normal except occasionally slight albumin. Blood count (forty-eight hours before operation): Hemoglobin, 83%; red blood cells, 5,520,000; leukocytes, 15,600; neutrophils, 18%; lymphocytes, 81%; mononuclears, 1%. Coagulation and bleeding time: Normal. Wassermann and Kahn: Negative.

*Operation.*—Repair of complete cleft of lip, right side.

*Anesthetic Time.*—One hour, twenty-five minutes.

*Operating Time.*—One hour, twenty-three minutes.

Details of a lip operation are included here to illustrate a typical case which may react to any operation.

"During operation, child reacted badly to a light degree of ether and oxygen anesthesia. Respirations shallow and jerky at times."

At completion of operation, 150 c.c. glucose given; patient's body seemed hot. Rectal temperature, taken on operating table, 108. Pulse 176. Respiration: arrhythmic. Patient was returned to room and given sponge bath and digalin.

Temperature before operation was 98. After operation, temperature ranged from 105.8, thirty minutes after operation, to 108, and was 107, thirty minutes before death eight and one-half hours after operation.

Temperature for two weeks prior to operation had ranged from 99 to 100 without known cause, but had returned to normal.

The important autopsy findings: A large tumor in apical region of heart, reported as primary rhabdomyoma. In the duodenum and the terminal portion of the ileum were closely set multiple areas of lymphoid hyperplasia. Enlargement of mesenteric glands, the largest measuring 12 x 7 mm. Congestion and edema of lungs but no pneumonia.

These were the main points of interest in this case of general lymph adenopathy. We had a patient, not normal in a general way, with variation in frequent blood counts and temperature range; yet with no outstanding contra-indication for operation unless the blood picture would have been considered such.

The blood picture in this child was abnormal. There was a definite leucocytosis. The lymphocyte count was 30 percent higher

than the normal upper limit for this age, the neutrophils 22 percent lower, and the mononuclear count definitely depressed. This picture would indicate that further laboratory and clinical study should be made to definitely determine the cause for these abnormalities.

In accounting for high temperature deaths in babies soon after operation, we must not overlook two possibilities; first, some interference with the normal action of the heat centers, second, reaction to the sudden injection of tissue proteins into the circulation. If the baby had a high protein sensitivity, there might be enough autogenous protein liberated by the normal amount of surgical trauma to produce the effect. If this is true, then a careful program of sensitization tests to food and bacteria should be carried out, and a careful history sought of any form of allergy in the parents.

It has been interesting to observe in our series that the patients having an enlarged thymus shadow and having had several x-ray treatments have not shown this post-operative reaction. Neither have they shown a visible tendency to hyperplasia of the lymphoid structure. This patient had a normal thymus shadow, and the gland showed no significant changes on microscopic examination, but there was a generalized lymphoid hyperplasia.

Wiseman<sup>1</sup> has shown in experiment with chronic foreign protein injections that "the thymus does not take part in the general hyperplasia of the lymphatic tissue which results. Repeated parenteral injections by various routes in a series of twelve rabbits caused an increase of lymphocytes in the peripheral blood varying from 23 percent to 139 percent. It seems probable that the degree of response is conditioned upon the type of protein used. At autopsy, the lymph nodes and spleen showed hyperplastic changes. The thymus did not participate in the hyperplasia."

This tends to disprove the long held idea that if there is an "enlarged thymus" there is likely to be more or less generalized and associated lymphoid hyperplasia. In speaking here of "enlarged thymus" and of the syndrome "Status Thymico-Lymphaticus," we are aware of the great diversity of opinion as to the existence of either as a pathologic entity even though the terms do occur in literature.

It appears that we must rule out allergy in these patients, and we should have been warned by the reaction following the first operation that any subsequent surgery might be followed by a more severe reaction. In the first operation there was less raw area of tissue exposed than at the second, and less trauma. Had more extensive surgical work been done in the first place, a fatality might have re-



sulted. Again the effect of the first operation may have been to increase the previous sensitivity to a point where even a minor surgical procedure would then have been fatal.

One naturally wonders whether some of these cases may not logically belong in the same category with those of sudden post-operative high-temperature deaths recently described by Connell,<sup>2</sup> who states, "An explanation based upon chemical, biochemical or metabolic disturbances seems much more probable than one depending upon microbic or bacterial infection." In his summary he says, "Rapid high-temperature deaths follow operations upon other than the biliary tract, probably more frequently than is supposed. One would seem justified in suggesting that the fatal syndrome is perhaps not hepatic but extrahepatic with possibly an important cerebral factor."

Connell,<sup>3</sup> Eiss,<sup>4</sup> and Shearer<sup>5</sup> had previously discussed the specific relationship of hepatic surgery to post-operative hyperpyrexia.

That the surgeon should be observant of the disease of the ear, nose, and throat, is evidenced by the frequent inflammatory involvement of these parts in this particular class of patients, especially prior to and during the surgical period.

The middle ear and maxillary sinus are often the seat of acute inflammations or infections. Especially is this so during the months of varying cold temperature and unsettled atmospheric conditions. We have frequently found, in a baby with excessive nasal discharge from the middle meatus, an unsuspected maxillary sinus involvement. This holds the infant back in a normal weight curve increase, causes intestinal disturbance as evidenced by abnormal stools and changes in the blood count. Of course, to close a palate cleft in the presence of this symptom complex is only to invite trouble, including infection of the raw surface of the palate.

In these patients with the oral and nasal cavities abnormally connected, often with both middle meati and respiratory fissures wide open to the oral cavity, the small percentage of ear and sinus disease is remarkable. Malnutrition, dehydration, improper feeding methods, etc., together with, of course, the acute exanthemata, seem to be the greater factors favoring infection of these parts.

The inhibitory action of the saliva and nasal secretion on bacterial growth must play a very important part in these patients who go through without infection. Since many of these babies if not closely watched put their hands, toys, toes, clothing and various other things in their mouths, they must carry bacteria into the mouth and nasal cavity.

The usual procedures employed in the treatment of inflammation or infection of the ears and nose may of necessity have to be varied in these patients to secure satisfactory results. Even after a successful closure of the cleft and after the patient is grown, he often presents a problem in the endo-nasal treatment of affections of the ear and nose because of the intranasal deformity. Particularly is this so in patients developing sinus disease, or tubo-tympanic involvement, whether suppurative or hypertrophic.

No rhinologist sees many of these patients with complications in the course of his practice and after a few years most of them are lost to the original operator.

Infections of the ears and sinuses occurred in 7 percent of the patients while they were under our observation. This does not include the frequent non-purulent reactions which may flare up and subside.

Acute tubo-tympanic congestion has been found in this series more frequently between the ages of three months and two years. Later on, while an acute inflammation can and does often occur, a chronic purulent otitis media or sinusitis or a hypertrophy with infection of the tonsils and adenoid more often is the complication at hand.

We realized some years ago the extreme importance of a rather detailed knowledge of the constant condition of these patients before operation. We found that the taking of certain tests at a certain time was a distinct advantage and in instances reduced the hospital days of the patient. To facilitate a more certain preparation for operation and to eliminate the possible omission of an important order, the following list of "standing orders" (with some subsequent changes) was adopted for the hospital several years ago. As in the case of *all* "standing orders," flexibility and variation for individual patients must be allowed and made.

#### CARE OF CLEFT LIP AND PALATE CASES

(Should be read by Resident and Intern before seeing any case. Charge Nurse should be thoroughly familiar with all of these orders.)

##### *Pre-operative*

1. Careful physical examination including ear, nose, and throat and a signed written report.
2. Intern to question family carefully as to recent illness or exposure to communicable disease or infection and family tendencies to disease; later charge nurse to question because family members may forget or refuse to tell the doctor of recent exposure to infectious disease, family tendencies towards such as a hemophilia and others that would contraindicate operation.

3. No case will be operated upon with acute inflammation of upper or lower respiratory tract or ears. Patient may show signs after examination indicated in Section No. 1 has been made. (Symptoms may develop twelve hours after this examination.)
4. Laboratory—for all ages. Throat culture on admission, repeated twenty-four hours before the operation. Wassermann and Kahn tests.
 

Blood count (complete). Hemoglobin. Coagulation time. Bleeding time. Urinalysis.	}	Report of <i>these</i> tests to be on chart within twenty-four hours after admission.
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- Vaginal swabs on all females, taken by charge nurse. X-ray of thymus on *all* children six years of age and under within twenty-four hours after admission.
5. Soda bicarbonate (or other alkali) q. s. P. R. N. twenty-four hours before operation to produce alkaline urine.
6. Skin tests for milk, eggs, and bacteria (on order).
7. No cathartics. Enema at 6 A.M. for patients six years or under on day of operation. Enema night before operation *and* morning of operation for patients six years or older.
8. Push fluids—fruit juice (orange) according to age up to six hours before operation. *Nothing after. Caution relatives not to feed or give fluids to patient.*
9. Patients two years and under—200 c.c. (or more according to age) of normal saline intraperitoneally one hour before operation. This solution is to be made from triple distilled water.
10. Temperature one-half hour before operation—*Important.*
11. Before sending to operating room, cleanse operative area and nostrils with boric acid swabs.
12. *Proportionate dose* (hypodermic) of atropine one-half hour before operation. (Be sure as possible of time of operation.)
13. Keep family from going to the operating room, if possible.
14. Send patient to operating room *warm* and well wrapped in a blanket, and *only* when anesthetic is to be started.
15. No infant operated upon:
  - (a) *Unless* on modified cow's milk diet (or mother's milk).
  - (b) *Unless* normal stools.
  - (c) *Unless* gaining weight—weigh daily.
  - (d) *If* regurgitating feedings.
  - (e) *Unless* "seems good" to pediatric nurse.

*Pre-operative Iodin Therapy*  
(On order by the surgeon)

According to age, suitable form of iodine in maximum dosage from time of hospitalization.

Adults: Lugol—10 to 15 drops a day.

Children: From four to six years of age: Iodid of iron or syrup of hydriodic acid (one-half to one teaspoon a day). If necessary—oidin (chocolate-coated tablets).

Babies: Syrup of hydriodic acid (10 to 15 drops a day).

## CARE OF CLEFT LIP AND PALATE CASES

*Post-operative*

(For babies and children—special orders for adults)

1. See that patient has dry clothing. May wrap in dry warm sheet and warm blanket and use hot-water bottles. *Have a warm bed.* Do not chill patients in changing garments or wrappings.
2. Watch respiration; that is, see that patient gets easy free breathing. If necessary, call interne.
3. Put cuffs on arms.
4. See that freshly prepared 6 percent solution of glucose is returned with patient from the operating room or ready when patient is returned from operating room. Have ready for glucose solution sterile bottle (2-4 oz.) (with sterile rubber-covered medicine dropper tip) contained in hot-water container.
5. When patient reacts and can swallow, give glucose solution (warm) carefully, P. R. N., ad. lib. to quiet and for twelve hours thereafter.
6. Feeding to be ordered by pediatrician and *before* the operation.
7. If patient shows signs of shock or unnatural post-operative signs, *call interne and surgeon. Don't wait.*
8. If patient is not quieted by glucose and is unusually restless and crying, give codein by hypodermic (by interne's orders) for three doses. After that, the needed dose may be repeated every four hours if required. *Never give any narcotic until the effect of the anesthetic has passed off completely.*

We know that a patient may be rendered to some degree unfit for operation by "over-preparation" but since the above system has been used (ten years), there has been a marked decrease in post-operative reaction and fatalities. Our chief worry at present is with the type of patient mentioned above in which the severe temperature, pulse, and respiratory reaction unexpectedly occurs.

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## SURGERY

The objective striven for in the surgical treatment of cleft palate is to secure union of the cleft borders in such a way that as nearly as possible normal function of the affected parts will result. To secure satisfactory function in the acts of deglutition, respiration, and speech in some is difficult even when the operation is done at the proper age. The parts which are so important in the acts of swallowing and respiration should be very carefully handled during operation and accurately and correctly approximated to secure, so far as possible, complete function. However, many cleft palate patients when they are older are more concerned about the speech element than either of the other functions of the palate. In other words, defective speech greatly influences the psychology of cleft palate patients. This psychological factor in these patients must not be overlooked, but the health and development of the individual should be our chief consideration. Straith<sup>1</sup> says, and he has many authors to substantiate his statement, "The psychological effects of congenital abnormalities begin to manifest themselves early in childhood."

We feel that too much stress cannot be made on the proper time for operation of the particular type of cleft at hand.

In our experience and observation of the work of others, surgery of the hard and soft palate, if begun before the proper age, usually results in partial or complete failure. Failure to secure primary union produces scar or loss of tissue so that the subsequent operation is difficult and the final restoration never so satisfactory as a successfully performed first operation. The over-ambitious or inexperienced surgeon does by injudicious surgery often permanently destroy a certain amount of function of these parts.

On the other hand, it is well known that surgery instituted several years after the proper age is of necessity more radical, and certainly cannot give the patient as good deglutitive, respiratory, or phonetic function as when done earlier, although it may, by some methods, secure a very good closure.

Failure to secure primary union in a satisfactory way is usually the result of ignoring certain facts which should be well known to the surgeon essaying to do this particular type of surgery. We say this because we see so frequently demonstrated the truths that:

1. One cannot successfully operate on the physically unfit.
2. One cannot satisfactorily operate at the wrong age.
3. One cannot succeed and ignore surgical principles.
4. Satisfactory results cannot be had with improper aftercare.

Brophy<sup>2</sup> in his last book on Cleft Lip and Palate lists fifteen different varieties of cleft palate. We have observed all types mentioned by him except number fifteen which is "Cleft only of the alveolar process anterior to the maxillae due to the non-development or absence of the premaxillae. Such cases are usually accompanied by cleft lip in the median line—inoperable." These types are rare, and many of the others occur only occasionally.

For the present purpose, it will be sufficient to classify all cases as one of two general types involving different surgical principles:

1. Cleft of soft and hard palate.
2. Cleft of the alveolar process.

In dealing with clefts involving only the soft palate or extending into the hard palate to varying degrees, the entire cleft is closed at one operation.

In our experience, closure of these clefts between the ages of twelve to eighteen months has given the most satisfactory results. No doubt if the cleft could be closed soon after birth and the united palate grow and develop with the growing baby, the ultimate function would be better, but for obvious reasons, we have not considered this plan. By the time the baby is twelve months old, we have a record of the general physical condition during this period and a good idea of fitness for operation. We have had ample time to study the renal and vascular systems. Blood studies give us much necessary information. The study of the endocrin system in infants presents a problem unless the endocrinopathy is one of extreme type. We feel, as before mentioned, that the baby with a disturbance of this function mild enough in form to escape our present day method examination, yet severe enough to manifest itself following surgery or other shock, is a very dangerous patient for operation.

At the fourteenth month, the soft tissues of the hard palate have developed sufficient thickness to withstand careful manipulation without rupture. The blood supply is adequate to facilitate good union. The soft palate has developed into a real muscular structure of sufficient size for easy surgical work. All these factors, in our experience, favor this age for operation on the soft- and hard-palate clefts.

The principle of the technic used is that of Brophy,<sup>3</sup> with some changes favorable to our way of operation.

If the cleft extends into the hard palate, the operation is begun by elevating the flaps of this structure, first, sufficiently to allow easy approximation. The periosteum is included in the flap. The soft palate is then detached from the posterior border of the horizontal

part of the palatine bones sufficiently to allow approximation. The borders of the entire cleft are then denuded of mucous membrane for suturing. The muscle tissue at the borders of the soft palate is then well exposed for approximation. At this stage, two No. 22 gauge silver wire stay sutures are introduced through the soft palate from the oral surface on one side and brought out through the opposite half of the structure, to be united on lead plates at the completion of the operation. These are only to oppose the action of the tensor palati muscle and prevent the soft palate suture line from opening under the strong contraction of this muscle. They are not used to draw together the cleft borders, and the tension must be correct to prevent injury to the muscle.

The entire cleft border is then closed with horsehair sutures and the operation ended. Careful attention is given to the uvula, as parents invariably comment on a well or poorly formed uvula, rather than upon its usefulness in the structure of the new palate.

By using the small sized wire fastened over the lead plates to prevent cutting, there is little or no injury to the muscle. When the palate is not contracting, there is no tension on the muscle. By this means, we have not found it necessary to use lateral incisions or any form of detachment of the muscles from their lateral anchorage.

In reference to the treatment of clefts of the alveolar process, operators have divided themselves into two main groups, one advocating closure and wiring of the parts to secure union; the other doing no surgery on the arch but closing the lip over the cleft, depending on the pressure of the closed lip to approximate the bones without union.

In our practice, the alveolar cleft is closed between the second and third month of age and held in position by silver wire, the soft tissue closed with horsehair. At this age, the bones are flexible and are not fractured. The technique varies here with the problem at hand.

If the cleft is a bilateral complete one with protruding premaxillary bone and if the patient can be operated on at the proper age, Brophy's technic<sup>4</sup> or some modification of it is used. In a personal communication from Prof. W. L. Shearer of Omaha, Nebraska dated August 27, 1934, he says, "I carry the Brophy operation through in its entirety in all cases where there is cleft of the alveolar arch." In older patients, the methods of Davis<sup>5</sup> and Shearer<sup>6</sup> have proven very satisfactory.

Bad results may follow the improper application of the technic described by the above-mentioned experienced operators in their

writing. The surgical principles and procedures described by them must be closely followed if results similar to theirs are to be obtained. It is a mistake sometimes made by those unfamiliar with this work to read a description of the operation and then to hope to accomplish the same results without having even seen the originator demonstrate the technic.

In this method also, the careful placing of the wires and the absence of too much tension is a most important step. I have never seen a troublesome hemorrhage or bone necrosis as a complication of this operation. We feel that a united arch is a part of an ideal restoration and more satisfactory later than one left ununited.

The patient with a cleft of the hard and soft palate who has been previously and unsuccessfully operated on *always* presents a problem to the operator. The patient with an improperly treated or ununited alveolar arch presents later a problem to the orthodontist, and the deformity is a source of personal discomfort and disadvantage. It has been our observation that the permanent upper front teeth in patients with a cleft of the alveolar arch are nearly always irregular with or without operation. We have seen a number of adults who have had no operation; their upper anterior teeth invariably have been irregular or defective. It is supposed that the same factor which causes the cleft, whatever that may be, may have been the cause of the irregular dentition.

As is the effect when placed too far back, the premaxillary segment, if set too far forward, causes a very noticeable deformity in the way of a receding or protruding upper lip and improper occlusion of the upper and lower teeth. The permanent upper teeth are irregular and often defective in every case of double complete cleft. They are especially so if no operation is performed; they are somewhat irregular with any technic used, and can be made worse, of course, by the improper use of wires.

In reference to the proper occlusion between the upper and lower teeth, Dr. Clair L. Straith of Detroit, Michigan, in a personal communication of August 18, 1934, says, "I have seen other cleft palates that have never had a Brophy operation and where the lower teeth occlude outside the uppers to give the same appearance that one sometimes sees in children who have been operated on by the Brophy method."

Following is an arrangement of our cases showing the age at operation, type of cleft, and number of each operated and the mortality.



Age	Number of cases operated— 318	Mortality				
		Number of cases				Total percentage
		Hard and soft palate	Per-cent	Alveolar arch	Per-cent	
3 months to	Arch 72 Palate 2	0	0	2	2.77	2.72
6 months	Total 74					
6 months to	Arch 23 Palate 101	1	0.99	1	4.34	1.61
24 months	Total 124					
2 years to	Arch 3 Palate 43	2	4.65	0	0	4.35
6 years	Total 46					
6 years to	Arch 10 Palate 49	0	0	0	0	0
12 years	Total 59					
12 years to	Arch 1 Palate 14	1	7.14	0	0	6.66
37 years	Total 15					

Heredity, 15% ; ear and sinus infections, 7% ; other deformities, 4%.

Our low mortality rate we attribute to a very painstaking pre-operative examination and preparation.

After the death of a patient we have sometimes felt that it was a mistake to attempt the operation, but in every such case the operation was performed with the advice and approval of the pediatrician and other consultants.

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## SPEECH ELEMENT

As before stated, the speech element concerns the patient more than the other functions of the palate after the self-conscious age is reached. Usually it is the main concern of the parents when the baby is first operated upon. We feel strongly that the palate cleft should be closed before or at the time of the onset of speech.

Hudson-Makuen<sup>1</sup> says, "The child begins to build up speech impressions and habits early in the second year, and the more serious forms of speech defects are those that are acquired during this formative period."

Russell<sup>2</sup> and many other authors<sup>3, 4, 5</sup> insist that the auditory patterns are established and even many speech sounds, such as *m*, *p*, *b*, *d*, *t*, the vowels, etc., are already well practiced before the middle of the first year.

As Wundt puts it: "Naturally all three steps pertain to speech development in its widest sense . . . first, crying sounds of vowel character, *ah*, *o*, *oo*, to the sixth week . . . ; second, from the seventh week to the end of the first and even second year in even normal children articulated sounds without thought content . . . characterized by the rapidly developing number of sound articulations . . . especially in the seventh to eighth month . . . first the vowels, then the consonants . . . ; third, thought content speech (P. R. 283-286)"

Most children with cleft palate, if operated on early, begin to talk at the usual age; if not they may be retarded and will not make the attempt until later. Speech is learned through the auditory, visual, and kinesthetic senses. The child attempts to reproduce phonetically the auditory and visual impressions from the speech used around him. The normal child, by checking his own phonetic output through these senses, in time learns to talk correctly. In other words, his audible results check with his speech impression and memory. This cannot be attained in the child with a defective peripheral speech organ.

When such a child checks his own imperfect attempts confusion results, and finally his faulty production is recorded in his speech center as his speech-directing mental pattern, which is, of course, incorrect; that is, he has incorrect speech impressions.

Hudson-Makuen<sup>1</sup> called this complex "cerebral impressions and peripheral habits." Once established, these impressions are difficult to break down.

Dorrance<sup>6</sup> says, "Patients with cleft palate are difficult individuals to teach new speech habits." These patients who have begun speech with a cleft palate have to be taught new speech like a new language so far as articulation is concerned.

Timbre pitch and intensity have to be regulated by correct breathing habits, as many of these patients, of necessity, waste much air in their attempts at speech. Exercises in correct breathing or use of the breath during speech must be employed.

This waste of air becomes a habit in complete clefts through failure to develop the three contact or stop points or positions which act as a resistance to the column of air during its passage through the peripheral speech organs. These points are: first, the dorsum of the tongue in contact with the soft palate and posterior pharyngeal wall; second, the tongue tip against the hard palate and teeth; third, the lips and teeth.

A soft palate which does not shut off the air column from the nasal cavity when it should disturbs certain speech sounds materially, such as *p*, *f*, *s*, *th*, etc., and the speech defect is called *rhinolalia aperta*.

Palatal adhesions restricting passage of the air, when it should not be restricted, as for *n*, *m*, and *ng*, cause a disturbed speech and voice similar to that heard from one who has a cold in the head. This difficulty is called *rhinolalia clausa*.

Since the soft palate or *velum palati* is a very active and the only movable part of the palate, it is the most concerned in speech. Therefore, it is our duty as palate surgeons to be most considerate in our manipulation and treatment of this muscle during operative procedures. It is composed of an intricate set of muscles, each with a special function. If a child in whom these muscles are separated by a congenital cleft can have them properly approximated at an early enough age with the least possible scar tissue or injury to the muscle body or tendons, he will stand a chance of developing almost normal speech under and following proper speech training.

This structure should receive as careful surgical consideration as the iris in an eye operation if good phonetic results are considered, because, as the iris regulates the light in vision, so does the soft palate regulate the air flow into the nasal cavity during speech. One of the outstanding speech defects of cleft palate patients is the failure of this air regulation.

Surgical faults which impair action are:

1. A short soft palate.
2. An improperly aligned union.
3. Unnecessary scar tissue.
4. Injury to muscle attachment and nerves.
5. Adhesions.
6. Injury during removal of tonsils and adenoids.

The effect of a short palate is obvious. If the borders of the soft palate are not approximated evenly, that is, if one segment is attached too high or "crimped" by badly placed sutures, the action of the palate will be impaired. Injury to the medial upper part of the superior constrictor during adenoid removal happens more often than is realized. In swallowing and in some speech production this part of the muscle contracts and forms a pad which meets the soft palate. A break in the continuity of this muscle impairs this function in proportion to the injury and secondary scar. The effect on speech is that of a short palate. Injury to the faucial pillars will shorten or stretch the palate and produce the same effect on speech and deglutition.

### RESONANCE

Resonance (Webster) is a prolongation or increase of sound due to the sympathetic vibrations of some body capable of moving in the proper period. Just as a series of small pulls at the right intervals will set a large bell to swinging, so resonance results from the repetition of small impulses which, when the bodies are "in tune," have a purely additive effect but otherwise tend to neutralize one another.

The idea has long been handed down that the nasal accessory sinuses aided in speech by adding "resonance" to the voice. Most of us have accepted this idea without question or investigation.

In securing resonance through the agency of air chambers, it is pointed out through experiment by Dr. G. Oscar Russell, Director of the Speech and Voice Clinic at Ohio State University,<sup>7</sup> that the openings of the chambers must be large enough to radiate the sound energy into space. He further states:<sup>7</sup> "We are talking of what artists, teachers of the voice, and others have designated as 'resonance.' A voice with good carrying power is said by them to be 'resonant.'"

More recently, he continues:<sup>8</sup> "Some oto-laryngologists and others have insisted that when singers develop sinus infections, the carrying power of their voices is materially reduced. Unfortunately such subjective impressions . . . without objective measurements of the exact amount of loudness variation . . . must be of questionable value. Furthermore, other factors may be involved; it is a well-known fact that the tissue in the neighborhood of the turbinates . . . tends to become congested and inflamed to such an extent that a radical muffling effect is inevitably brought to bear on any tone which normally is passed through the nose. Since a substantial part of the consonants such as *m*, *n*, etc., and some of the vowels regularly call



for an opening of these passages, their loudness must of course be affected, and any aria, song, or other connected word singing would thereby be radically modified. Furthermore, since the posterior part of the nasal passages would be open and the anterior closed, any tone which entered the cavity would be caught in a closed cavity muffler. We know that closed tubes of this type respond only to the odd numbered partials, and where loud vibrations are set up in them, a very disagreeable nasal quality results. . . . Anyone can demonstrate this fact for himself by pinching his nostrils while he intones a rapid succession of syllables in which a hummed *m* is coupled with various intoned vowels such as *mah-mah-mah*, or *me-me-me*. In all probability, however, he will find some vowels less disagreeable such as *moo-moo-moo*. The reason is probably to be sought in the fact that the high partials are practically absent in the *moo* . . . and strongly present in the *me*. In this case the sinuses are all still present, so the reduced loudness and bad quality cannot be blamed on them.

"If the sinus had any function at all, it might well be expected to produce exactly such a dissonant effect. For, it has but one small opening and no power of varying its capacity. Consequently it could by no means blend in harmonic relationship with the other elements in a complex tone which was always changing its fundamental pitch as it does in a scale or song. If such a sinus did serve as a 'resonator,' it would boom out whenever its pitch were encountered in a scale, and sink on all other pitches. The result would be hideous.

"In a demonstration before the Chicago Council of Teachers of Singing, June 12, 1934 . . . our apparatus for measuring the effective loudness and quality modification where the laryngeal tone was produced was set up: (1) in the cadaver; (2) in the cadaver head with one and then both antra opened up and removed; (3) with the frontal sinus removed; (4) with the sphenoidal sinus removed; (5) with the whole top of the head including all the nasal passages cut off and nothing but the buccal passage or megaphone left; (6) with the whole head removed. Since the air pressure and glottic tension was kept an absolute constant for all experiments, an exact experimental comparison of effects could thereby be obtained. . . .

"Practically no loudness change could be discovered by either the presence or removal of any of the sinuses. Nor could any quality differentiation be detected. However, when the whole top of the head was removed, including the nasal passages, the resultant tone was distinctly less mellow and far more blatant . . . and loud. . . . For in this case the glottal tone was concentrated through the mouth

functioning as a megaphone, flaring progressively from the small opening over the larynx to the lips with most of the superimposed mellowing modifiers removed. ”

All of this is leading up to the statement that the present-day theory of the nasal cavity and accessory nasal sinuses acting as “resonators” for the human voice is incorrect, and that they can serve at best, if at all, only as modifiers of the voice.

It would seem that that long assumed function of resonance is no longer to be ascribed to the sinuses for the following reasons: the small openings of the sinuses limit sufficient back-and-forth surge of air to adequately radiate sound waves into space; they are tuned to only one pitch; they are lined with a soft moist covering which would tend to the absorption and deadening of sound waves. This is indeed gratifying to the cleft-palate surgeon who has in so many instances been accused of not having restored the “nasal resonance” by his operation.

We know that in all patients with a double complete cleft palate, the nasal septum is always irregular after operation no matter what technic has been used. We know that most of these patients have hypertrophy or other irregularities of the turbinates (inferior) which limit the normal nasal breathing.

The disagreeable quality of the voice of a person having a marked nasal obstruction from one cause or another has been termed increased nasal resonance by many. If we discard the idea of this cavity as a resonator and consider the obstructed nasal cavity and possibly an inactive soft palate (making the naso-pharynx a dead cavity) as acting on the passage of sound waves, as an obstructive middle-ear disease does in hearing, we shall be in agreement with what seems now the demonstrated and objective scientific experimental evidence.

The waves, rebounding from the obstructed cavity or unclosed naso-pharynx into the oro-pharynx, mix with the other voice sounds and, by neutralizing some and modifying others, produce an unpleasant voice quality.

All of these factors, together with the change in our idea of “nasal resonance,” give us an explanation of some of the speech defects in cleft palate patients who have been carefully operated upon.

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### SUMMARY

1. Periodic observation of the patient from time of birth up to the time of operation has been a distinct advantage.

2. In addition to the usual preoperative examination made for surgical cases, other special ones must be made. This is particularly important in patients two years of age or younger. The results of the various examinations should be carefully correlated by the surgeon himself to look for isolated facts as elements of an unsuspected and possibly important contraindication to operation.

3. Special preparation of the patient for some time before operation reduces the post-operative reaction to a minimum.

4. Intelligent attentive nursing under the direction of a dependable supervisor, experienced in the care of this class of surgical patients, is absolutely necessary.

5. Early closure of clefts of the alveolar process and clefts of the hard and soft palate before the onset of speech is advisable.

6. Speech training should be begun soon after operation. It is a long process but much can be accomplished by a competent instructor if full cooperation can be secured.

### AUTOPSY

(Detailed Findings on Case Reported)

*General Description.*—The body is that of a well-developed white male baby nine months of age, measuring twenty-eight inches in length. The eyes are brown and the hair is light and thin. The jaws are firmly closed by rigor mortis and are not forced apart. The right side of the upper lip has been recently repaired in the process of closure of a harelip. The head appears to be slightly larger than normal but the contour remains regular and is not of sufficient size to be classed as a hydrocephalus. The posterior fontanelle is completely closed while the anterior fontanelle is large, depressed and ossified only about its margins. There is a marked post-mortem lividity but no special marks of identification.

*Thoracic Cavity.*—Upon opening the thorax, the cardiac area is seen to be of the usual size and position. The thymus is not enlarged, being instead somewhat small for a child of this age and upon removal being found to weigh 9 gms. Both lungs are entirely free and there is no increase in pleural fluid. The right lung is entirely crepitant but deeply congested throughout and in its lower lobe has somewhat the appearance of an early bronchial pneumonia. There are a few petechiae over its anterior pleural surface. The left lung is entirely similar. The trachea and bronchi are entirely free and open. Upon opening the pericardial sac, it is found to be smooth and glistening and to contain the usual amount of fluid. The heart and great vessels are in the normal position but the heart is observed to be apparently elongated and in the apical portion is of a peculiar whitish color. Upon removal of the heart, it is found to weigh 46 gms. and the apparent elongation of the heart, above noted, as seen to be in the longitudinal diameter which measures  $5\frac{1}{2}$  cms. from the atrioventricular groove to the apex on the anterior surface of the heart. Upon opening the heart, the right ventricle appears to be somewhat small and its cavity measures  $25 \times 22$  mm. The pulmonary and tricuspid valves are normal. The pulmonary valve measures 25 mm. in circumference and the tricuspid 45 mm. The myocardium of the right heart is of the usual thickness. Upon opening the left side of the heart, the left ventricle is found to be of normal size, its cavity measuring  $40 \times 26$  mm. The mitral and aortic valves are not pathologically noteworthy and the mitral valve measures 38 mm. in circumference and the aortic 30 mm. The foramen ovale is entirely closed and the ductus arteriosus is also obliterated. In the apex of the heart, occupying the position of the interventricular septum and extending into the myocardium at the apex of the left ventricle, is a hard mass which gives the heart the whitish color above noted in this region. Upon cut section, this hard mass is found to be a dense tumor measuring  $22 \times 25$  mm. The tumor is well circumscribed from the overlaid by a thin layer of grossly normal myocardium. The tumor is of a dense consistency, a whitish color, and presents fine interlacing bundles of a fibrous-like tissue. There is no evidence of degeneration. The coronary arteries are not pathologically noteworthy. The lymph nodes within the arch of the aorta and also the peritracheal and peribronchial nodes are moderately hyperplastic.

*Abdominal Cavity.*—Upon opening the abdomen, the peritoneum is found to be smooth and glistening and there is no peritoneal fluid. The liver is of the usual size and upon cut section is slightly mottled. The gallbladder, pancreas and spleen are normal. The stomach is slightly distended with gas but upon opening is found to be empty and not noteworthy. The pylorus is patent and upon opening the duodenum, its mucosa is found to be studded with small, closely-set elevations which have the appearance of multiple areas of lymphoid hyperplasia. Upon opening the remainder of the intestinal tract, a similar but less marked area of multiple elevations is found in the terminal portion of the ileum but the remainder of the intestinal tract is normal. The mesenteric lymph nodes are diffusely enlarged, the largest measuring  $12 \times 7$  mm., and being diffusely congested. Upon dissection of the left kidney, the left ureter is found to be dilated to an average diameter of 4 mm. and upon removal and cut section of the left kidney, its pelvis is also found to be dilated and filled with a turbid urine. The kidney itself is not especially remarkable upon cut



section. The right ureter is also dilated as is also the right kidney pelvis but to a less extent than the corresponding structures on the left. Both adrenal glands are normal. The urinary bladder is moderately distended with a turbid urine.

*Cranial Cavity.*—Upon dissection of the scalp, the various suture lines are found to present an unusual degree of ossification such that it is necessary to use a bone saw to open the skull. The skull is otherwise not noteworthy. Upon removal of the calvarium, the dura is found to be of the usual thickness and upon removal of the dura the hemispheres of the brain are somewhat congested but otherwise not noteworthy. The pia arachnoid membranes are not thickened. Upon removal of the brain, multiple cut sections fail to reveal any pathological changes aside from those of congestion which congestion is probably due to the length of time intervening between death and performing the autopsy. The ventricles are not dilated.

*Anatomical Diagnosis.*—1. Primary tumor of the heart. 2. Generalized lymph adenopathy. 3. Possible bronchial pneumonia. 4. Hydrocephrosis. 5. Recently repaired harelip and cleft palate.

*Microscopic Sections.*—Sections of both lungs show a marked congestion and edema but no evidence of pneumonia. The bronchi are free and open and the mucous glands in the sub-bronchial tissue are normal.

Sections of the duodenum show a marked hyperplasia of lymphoid tissue in the submucosal layer with displacement outward and erosion of the overlying mucosa.

The pancreas and liver are microscopically normal.

The spleen is moderately congested but otherwise not noteworthy.

The kidneys are congested but show no noteworthy pathological changes.

The mesenteric lymph nodes show a marked hyperplasia and are congested almost to the point of hemorrhage.

The thymus contains the usual amount of lymphoid cell infiltration and the Hassel's corpuscles are not prominent. The fibrous stroma is well marked.

Sections of the tumor in the heart show it to be composed of spindle to stellate forms of cells which somewhat interlace and contain large spaces which the ordinary stains are apparently empty. The nuclei are elongated and spindle shaped and moderately granular. The picture is that of a primary rhabdomyoma of the heart which apparently contains a large amount of fibrous tissue.

Sections made through the various levels of the medulla show the various cellular elements to be well preserved and in the normal relations. There is no increase in glial tissue or evidence of degeneration. Sections through the floor of the fourth ventricle show the cellular nuclei entirely normal. Sections taken through the mid-brain and lateral ventricles and nuclear regions are not pathologically noteworthy aside from some congestion. Sections through the cortical regions are also normal.

*Final Diagnosis.*—1. Primary rhabdomyoma of the heart. 2. Generalized lymphoid hyperplasia with the exception of the thymus. 3. Congestion and edema of the lungs. 4. Hydronephrosis. 5. Recently repaired harelip and cleft palate.

## NEUROLOGICAL ASPECTS OF FRONTAL LOBE ABSCESS\*

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The occurrence, in my practice, of two cases of abscess of the frontal lobe, one entirely without symptoms, though its presence was suspected and every effort made to establish its diagnosis before operation, roused my interest and led to an attempt to see how true was Gerber's statement (quoted by Leegaard<sup>12</sup>) that "the common symptom of frontal lobe abscess is its absence of symptoms." A search was made, therefore, through the records of the twenty-five local hospitals, and twenty cases of frontal lobe abscess which had never been reported in the literature were found; most of these had been discovered at operation on the frontal sinus, in exploratory proceedings for undiagnosed intracranial lesions, or at autopsy.

The records of the pathological department of the state university, covering a period of thirty-five years and 22,700 autopsies, were next checked; the reports found, however, had already been obtained from the hospital records examined.

The next step was a search of all available English and American literature for reports which might be those of frontal lobe abscess. Only cases were considered in which the presence of a solitary abscess had been definitely established, either by operation or post-mortem; many cases of multiple abscesses were rejected because of the element of confusion introduced into the neurological picture by such lesions. Two hundred and four cases fulfilling the above requirement were found.

These case reports were critically reviewed for neurological signs; the findings constitute this paper and will be discussed under the following headings:

- A. General discussion.
- B. Cranial nerves to extrinsic ocular muscles.
- C. Cranial nerves and sympathetic system to intrinsic ocular muscles.
- D. Optic nerve.
- E. Pyramidal tract.
- F. Motor cranial nerves not elsewhere discussed.

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\* Candidate's thesis.

- G. Sensory disturbances.
- H. Reflexes.
- I. Aphasia.
- J. Psychic changes.

#### A. GENERAL DISCUSSION

The diagnosis of frontal lobe abscess has always been attended with such difficulty and so many cases have been recognized only at the postmortem table that the tradition has come down in the literature that frontal lobe abscess is diagnosed by its absence of symptoms. Leegaard<sup>12</sup> remarks that "in these cases (frontal lobe abscess), as a rule, no focal symptoms are to be found . . . . In general, in making the diagnosis, we must be content with the etiology of the case (the sinusitis, combined with the more or less distinct signs of intracranial pressure. In this way diagnosis has in many cases been successfully made. But this nearly always demands a certain time for observation. And often the mode of procedure in the first place is to operate for the sinusitis. If the symptoms do not disappear, as has been expected, if they continue and increase, it gradually becomes clear that there exists something more than an inflammation in the frontal sinus and then finally the cerebral abscess is diagnosed. Such a state of affairs is so typical that on reading through the history of cases we find these conditions recurring again and again almost with the regularity of a law." Skillern<sup>21</sup> bore witness to this truth when he said, "I do not recall a case where the operator opened up the frontal sinus and drained an abscess—I mean a blind brain abscess in the frontal lobes—that was diagnosed primarily."

Absence of symptoms is more characteristic of the right than of the left frontal lobe, owing to the presence of speech centers in the latter; Neumann<sup>17</sup> recognizes this difference when he observes, in speaking of otitic brain abscess, that "the prognosis for right-sided abscesses is better, since we have not to wait for the aphasia, the characteristic symptom of left-sided abscess. On the contrary, we are obliged to make our diagnosis on the basis of general symptoms and, therefore, make an exploratory brain puncture much earlier than in cases of left-sided abscess with aphasia." If this be true of otitic temporal lobe abscess it should hold to a certain extent for abscess of the frontal lobes.

Even as Neumann<sup>17</sup> stresses the importance of general signs of brain abscess, so Macewen<sup>13</sup> called attention to the value of local symptoms: "When symptoms were present pointing to a lesion in one of the recognized cerebral centers, it was better to depend on

them as guides, at least to the encephalitis surrounding the abscess, than to trust to the indications afforded by the scalp or skull injury."

The need and value of localizing signs, always great in any intracranial lesion, become doubly so in the presence of bilateral frontal sinusitis; confusion arises from the fact that the sinusitis may produce symptoms closely resembling those of the complication, so that lateralization of the latter becomes difficult or impossible; in such a situation the faintest localizing sign becomes important.

Experience teaches that frontal lobe abscess may attain great size without symptoms; Leegaard<sup>12</sup> observes that "we can with fairly great certainty count upon the cerebral abscess not being far distant from the cerebral wall of the frontal sinus. It is, therefore, directly behind this latter that we must look for the abscess. These abscesses, by the time we are in a position to diagnose them with any approach to certainty, have as a rule attained pretty large dimensions." Halstead and Vaughan<sup>10</sup> thought that these abscesses frequently occur low in the second and third frontal gyrus and do not, therefore, involve motor tracts and centers as frequently as do tumors, which are apt to be higher up and further back; abscess has a tendency to be to the front and median line.

Abscess of the frontal lobe produces neurological signs by pressure upon or destruction of nerve centers and tracts. Destruction of tissue by inflammation is always a local process; whether or not localizing symptoms result depends on the presence of structures (centers and tracts) in the area involved, destruction of which is attended by such symptoms. Increased intracranial pressure, caused by the abscess itself, or by its attending edema, congestion, or encephalitis, may be direct or transmitted, localized or generalized, of slight or of great degree. Direct pressure is exerted upon contiguous structures, is localized, and produces localizing symptoms if important structures are involved, *e.g.*, pressure upon the precentral gyrus with resultant Jacksonian attacks, pressure upon the olfactory nerve with resultant anosmia. Transmitted pressure may manifest itself by symptoms arising from involvement of structures at a distance, *e.g.*, hemiplegia from an abscess in the frontal pole of the frontal lobe. Direct or transmitted pressure may be of slight degree, causing symptoms of irritation, such as twitching, spasm, motor weakness (motor nerves), or paresthesia (sensory nerves); or of great degree, causing convulsions, paralysis, or anesthesia.

Increased size of the frontal lobe, evidenced by tense dura and flattened gyri, may cause mass displacement of the cerebrum with its resultant compression against the cranial walls, especially the floor; the medulla may be compressed as it passes through the tentorium.



Absence of symptoms may be attributed, not only to the fact that important centers and tracts are not involved, but also to the fact that the flow of cerebrospinal fluid is not obstructed; absence of firm dural attachments, while permitting displacement of the cerebrum, may also prevent undue stress from such displacement.

### B. EXTRINSIC OCULAR MUSCLES

Paresis or paralysis, much less commonly spasm, of the extrinsic ocular muscles is seen in increased intracranial pressure and hence in large frontal lobe abscess; the opinion is quite generally expressed that such paralysis has little localizing significance and indicates increased pressure only. Eagleton<sup>6</sup> makes the statement that oculomotor paralysis has little, if any, localizing value, that trochlear paralysis localizes the lesion in the posterior fossa near the midline, and that abducens paralysis stands between the two in localizing importance. Fuchs<sup>8</sup> states that paralysis of the individual trochlear or abducens nerve has little or no localizing value, that involvement of the oculomotor nerve may permit of some localization by studying the distribution of the lesion among its branches; Coleman<sup>6</sup> feels that ocular palsies are of little importance.

Involvement of the extrinsic ocular muscles in increased intracranial pressure is easily understood when one considers the anatomy of their nerve supply. The nuclei of the oculomotor and trochlear nerves are in close proximity in the midbrain beneath the corpora quadrigemina and the aqueduct of Sylvius; the abducens nucleus is a centimeter lower down in the pons, close to the genu of the facial nerve under the floor of the fourth ventricle. This entire area is vulnerable to increased pressure. The nerves themselves are frequently involved in their intracerebral courses from their nuclei, and in their intracranial courses from their points of superficial origin in the brain stem to their exits from the skull through the superior orbital fissure. The exits from the brain of the oculomotor and abducens nerves are near the pyramidal system; the abducens may actually pass through this tract and the trochlear nerve curves round it. These relations explain the frequent association of motor disturbances of the extrinsic ocular muscles with those of the contralateral side of the body.

The cranial nerves, through their nuclei and the pyramidal tracts, have bilateral representation in the motor cortex, mostly contralateral, as a result of which a unilateral lesion in the upper motor neuron does not usually cause paralysis, a wise provision on the part of nature to protect such vital functions as swallowing and breathing.

It is not necessary, therefore, to consider the supranuclear tracts and the motor cortex in attempting an explanation of the ocular palsies occurring in frontal lobe abscess; they must be peripheral, in the intracerebral or intracranial portion of the nerve.

These nerves may be divided into zones to facilitate discussion of the localizing value of their lesions. Whitnall<sup>23</sup> suggests that any orderly treatment consider successively the nucleus of origin, the intracerebral course, the superficial origin, the intracranial course, the path through the cavernous sinus, the orbital course, and the final distribution (muscles). Fuchs<sup>8</sup> divides paralyzes into cerebral (brain), peripheral (peripheral nerves), and muscular (muscles), or intracranial and orbital. Finer division of the intracranial group would include cortical, supranuclear, nuclear, and fascicular (nerve trunk) lesions.

#### I. OCULOMOTOR NERVE

The oculomotor nucleus consists of several paired and one unpaired group of ganglia. The former constitute the large-celled lateral chief nucleus of Whitnall, supplying the extrinsic muscles of the globe, and are subdivided from before backward into smaller groups of cells supplying individual muscles, levator palpebræ superioris, rectus superior, rectus inferior, and obliquus inferior; the unpaired ganglia, composed of two smaller-celled nuclei in front (Edinger-Westphal nucleus), and one behind and below fused with its fellow of the opposite side to form a central mass, supply most of the intrinsic muscles of the eye (sphincter of the iris and the ciliary muscle).

It is generally agreed that fibers destined for the levator palpebræ superioris and rectus superior are entirely uncrossed and those to the rectus inferior entirely crossed. Disagreement exists, however, as to the composition of the nerves to the rectus internus and obliquus inferior. Fuchs<sup>8</sup> represents the fibers of the rectus internus as entirely uncrossed, while Whitnall<sup>23</sup> considers them to be both crossed and uncrossed. Fuchs represents the fibers of the oblique inferior as entirely crossed, while Whitnall considers them to be both crossed and uncrossed. This division of the oculomotor nucleus and disposition of its fibers has not been certainly demonstrated in man, but is thought to hold for apes.

From its superficial origin in the oculomotor sulcus above the pons the nerve runs a course of 25 mm. in the posterior fossa to enter the cavernous sinus, through which it courses to pass into the orbit through the superior orbital fissure. Paralysis may result from injury at any point in its entire course.

Table 1 summarizes the lesions of the oculomotor nerve.

The terms ipsilateral and contralateral, as used throughout this study, refer to the side of the cerebral abscess.

TABLE 1.—OCULOMOTOR LESIONS

Nerve	Number of cases	Other ipsilateral oculomotor lesions				Abducens paralyses		Facial paralyses		Ipsilateral pyramidal tract	Ipsilateral complete ext. ophthalmoplegia	Orbital disease
		Dilated pupil	Superior rectus paralysis	Internal rectus paralysis	Complete oculomotor paralysis	Ipsilateral	Bilateral	Ipsilateral	Contralateral			
Levator palpebrae superioris..	16	6	..	..	1	2	1	..	4	8	2	2
Superior rectus .....	4	1	..	1	..	..	..	1	1	..	..	3
Internal rectus .....	2	1	1	..	..	..	..	..	..	..	..	..
Total .....	22	8	1	1	1	2	1	1	5	8	2	5

A lesion involving the intracranial portion of the oculomotor nerve might logically be expected to involve other nerves in the immediate vicinity, such as the trochlear; in three cases in which such a lesion was probable (two of complete external ophthalmoplegia and one of complete oculomotor paralysis) there was one example of paralysis of the ipsilateral trochlear nerve. The contralateral facial nerve was involved once, the ipsilateral pyramidal tract twice.

Again, it is difficult to picture the site of any isolated lesion, either in the intracranial course or nucleus of the oculomotor nerve, which could involve only the fibers destined for an individual muscle. Yet, eliminating from the above group of twenty-two cases of oculomotor involvement the one case of total oculomotor paralysis, twenty-one are left in which only partial involvement of this nerve was present; two of these were cases of total external ophthalmoplegia.

#### SUMMARY OF OCULOMOTOR LESIONS

Involvement of the oculomotor nerve was present in 22 of 204 cases of frontal lobe abscess. In only one case was the paralysis total. In all others it was partial and manifested most frequently, in the order named, by paralysis of the levator palpebrae superioris, rectus superior, and rectus internus.

Other neurological signs associated with lesions of the oculomotor nerve were contralateral motor disturbances of the body, due to involvement of the ipsilateral pyramidal tract above its decussa-

tion, dilation of the ipsilateral pupil, and paresis of the contralateral facial nerve.

Difficulty arises if one attempts to localize the site of the lesion in this group of twenty-two cases; Fuchs<sup>8</sup> remarks that complete oculomotor paralysis almost certainly involves the intracranial course of the nerve proximal to the superior orbital fissure; it could scarcely be nuclear, owing to the fact that the internal rectus and inferior oblique muscles are partially, and the interior rectus muscle wholly, activated by crossed innervation and would, therefore, in an ipsilateral nuclear lesion, receive stimulation from the contralateral normal nucleus.

Consideration of these facts permits only the conclusion that lesions of the oculomotor nerve, alone or associated with involvement of other nerves to extrinsic ocular muscles or with involvement of the pyramidal tract or facial nerve, have no exact localizing value, but point only in a general way to the middle or posterior fossa of the skull as the probable site of the lesion. They have some laterilizing value in that they occur more frequently on the side of the frontal lobe abscess than on the opposite side.

## II. TROCHLEAR NERVE

The trochlear nerve's intracranial course is long (40 mm.), largely because it emerges from the posterior side of the brain stem about which it swings to join the oculomotor and abducens nerves; it passes through the cavernous sinus and enters the orbit through the superior orbital fissure to supply the superior oblique muscle. Trochlear paralysis results in inability to depress and intort the eye. The nerve is rarely affected alone; only one such involvement occurred in this study.

Eagleton<sup>1</sup> states that trochlear paralysis localizes the lesion in the posterior fossa near the midline. Its common involvement with other nerves to extrinsic ocular muscles carries with it the localizing and lateralizing values attributed to lesions of those nerves.

## III. ABDUCENS NERVE

There were six cases of ipsilateral abducens paralysis, two of contralateral, and three of bilateral involvement, in 204 cases of frontal lobe abscess.

The statement regarding the lack of localizing value of paralysis of the extrinsic ocular muscles applies with especial force to abducens paralysis. It has a long intracranial course and is especially liable to trauma on account of its exposed position on the ventral surface



of the pons, where there is very little space between the body wall and the brain. In increased intracranial pressure it is apt to be compressed against the pons by the posterior cerebral branch of the basilar artery, which is pushed down against the inferior olive. Sixth nerve palsies, common in fracture of the base and in petrous tip suppuration, have, therefore, but little localizing value. The fibers are entirely uncrossed.

Table 2 records the lesions of the abducens nerve.

The occurrence of orbital disease in four of six cases of ipsilateral abducens palsy raises the question as to whether the lesion was orbital or intracranial; the presence, in one case, of involvement of the contralateral facial nerve and ipsilateral pyramidal tract, points to the middle or posterior fossa, rather than to the orbit, as the site of the lesion. There were no associated signs in the other three cases to indicate definitely whether the lesion was orbital or intracranial.

TABLE 2.—ABDUCENS LESIONS

Side	Number of cases	Oculomotor paralyses					Facial paralyses		Ipsilateral pyramidal tract	Orbital disease
		Ipsilateral		Contra-lateral	Bilateral		Ipsilateral	Contralateral		
		Ptosis	Fixed dilated pupil		Dilated pupils	Ptosis				
Ipsilateral .....	6	1*	..	..	..	..	..	1	1	4
Bilateral .....	3	..	3	2	..	3	..	3	3	1
Contralateral .....	2	1	..	..	2	..	2	..	..	..
Total .....	11	2	3	2	2	3	2	4	4	5

\* Probably orbital due to foreign body abscess.

One case presented an ipsilateral ptosis which was, however, probably peripheral in origin, inasmuch as there had been a foreign body orbital abscess with considerable lid trauma.

Ipsilateral abducens paralysis occurred twice without orbital disease; the absence of facial nerve involvement in these cases points more to the intracranial portion of the nerve than to its nucleus as the site of involvement because the nucleus of the abducens is in contact with the genu of the facial nerve and its disease is usually accompanied by disturbances in facial musculature. This is not always true, however, because pressure may not equally affect gray matter (nuclei) and white (fibers); its exertion, in non-destructive lesions,

on the facial colliculus (prominence on the floor of the fourth ventricle where the facial nerve turns around the nucleus of the abducens) might cause a lesion of one nerve and not of the other.

#### SUMMARY OF ABDUCENS LESIONS

Lesions of the abducens nerve were most frequently associated with involvement of the ipsilateral pyramidal tract, contralateral facial nerve, or oculomotor nerve of either side; unilateral lesions of the oculomotor nerve were most frequent on the ipsilateral side.

All cases presented definite evidence in fundi and reflexes of considerable increase in intracranial pressure and it is only logical to assume that this pressure affected the nerves in question on each side, either directly or indirectly.

The only significance which may, in cases of frontal lobe abscess, be attached with certainty to lesions of the abducens nerve is that of increased pressure in the middle or posterior cranial fossa.

Such lesions have some lateralizing value in that, when unilateral, they occur more frequently on the side of the cerebral abscess than on the side opposite the abscess.

#### SUMMARY OF PARALYSES OF EXTRINSIC OCULAR MUSCLES

Paralyses of extrinsic ocular muscles may be caused by disease of the orbit. Their occurrence, in cases of frontal lobe abscess not so complicated, is due to increased pressure in the middle or posterior fossa of the skull. Their frequent combination with motor disturbances in the field of either facial nerve or of the pyramidal tract, usually of the same side, also points to the pons or midbrain as the site of the lesion.

The more frequent occurrence of unilateral ocular palsies on the side of the cerebral abscess is of some lateralizing value which, however, is limited by the occurrence of such lesions on the side opposite the abscess.

Bilateral ocular palsies are indicative only of increased intracranial pressure and have no localizing value.

The nuclei and intracerebral portion of the oculomotor, trochlear, and abducens nerves are in such close proximity in the midbrain and pons and their intracranial segments on the base of the skull in such intimate relationship that increased intracranial pressure does not produce a neurological picture of distinctive design into which any exact localizing or lateralizing significance can be read.

The incidence of ocular palsies was not appreciably influenced by the presence of orbital disease.

## IV. CONJUGATE DEVIATION

Conjugate deviation of the eyes with or without associated turning of the head, either to the same or opposite side, is a phenomenon frequently mentioned in connection, not only with frontal lobe abscess, but also with other diseases, such as epilepsy. Halstead and Vaughan observed conjugate deviation of the head and eyes premonitory to Jacksonian epilepsy; Osler<sup>18</sup> and Atkinson<sup>1</sup> noted the same finding. In this series of cases conjugate deviation of the eyes was noted three times, twice to the side of the lesion and once to the opposite side. Conjugate deviation of the eyes and turning of the head to the side of the lesion was noted but once. In none of these three cases did the accompanying neurological findings form any characteristic picture.

Whether or not conjugate deviation of the eyes has any localizing value depends, of course, on its mechanism. This is to be found in the oculomotor, trochlear, and abducens nuclei, the posterior longitudinal bundle, and certain special centers which are thought to act as coordinating and distributing points for the impulses concerned in conjugate movements; the innervation of the recti muscles (the internal rectus of one side and the external rectus of the other), is especially important in lateral movements.

Ballance<sup>2</sup> states that the center concerned with movements of the head and eyes is located in the area between the posterior end of the second left frontal convolution (containing Charcot's motor graphic area) and the posterior end of the third left frontal convolution (Broca's speech center); he quotes Mills to the effect that the symptom complex of the region is motor agraphia, motor aphasia, fits, movements of the head and eyes, and psychic changes. Grinker<sup>9</sup> mentions not only this center, stating that it is probably that for voluntary movements of the head and eyes, but also a center in the occipital lobe, concerned chiefly with reflex changes in eye position, and adds that these centers are antagonistic, irritative lesions producing movement toward the opposite side and destructive lesions, movement toward the same side.

Riley<sup>20</sup> describes special coordinating centers for lateral and vertical conjugate eye movements and for convergence; a special center for divergence has not been demonstrated.

The center for the control of lateral conjugate movement is thought to be near but not identical with the abducens nucleus, sending fibers directly to the homolateral abducens nucleus and to the contralateral oculomotor nucleus through the posterior longitudinal bundles.

Spiegel,<sup>22</sup> in a recent paper, reports experimental work on cats which suggests strongly that the impulses from the frontal lobe motor centers for eye movements, after passing through the pyramidal tracts, enter the vestibular nuclei. Here they are redistributed to enter the ascending fiber tracts of the posterior longitudinal bundles of each side to reach the abducens and oculomotor nuclei. Riley<sup>20</sup> also mentions the rôle of the vestibular nuclei in lateral deviation, stating that impulses pass, chiefly from the nucleus of Dieters, directly across the tegmentum to enter the posterior longitudinal bundle of each side and end in the special centers controlling lateral deviation.

The superior colliculus, according to this author, may be considered the special center or redistribution point for impulses controlling vertical deviations; the mesial portion of that segment of the oculomotor nucleus innervating the internal rectus muscle, the nucleus of Perlia, is considered to be the special center for convergence.

In speaking of the importance of the posterior longitudinal bundle in conjugate movements Riley<sup>20</sup> says: "Its constituents number, not only the internuncial fibers passing between the various centers already mentioned and between the various nuclei of the oculomotor apparatus, but also the fibers that are concerned with the reflex activities of the eye movements, these being chiefly represented by the trigeminal, the acoustic, and the vestibular contributions. It also serves to transmit the impulses that are received from suprasegmental sources that control the associated, synergic, and voluntary control over the oculomotor movements."

Meyers,<sup>15</sup> in a recent paper, finds conjugate deviation of the eyes a valuable diagnostic sign, especially in unconscious patients or in cases in which no history can be elicited and observes that it is less frequently noted in tumor than in abscess because the slow development of the tumor permits compensatory action of other centers which corrects the deviation; shifting and stretching of the entire brain also minimizes the effect of tumor. In a rapidly developing abscess the persistence of conjugate deviation in increasing intensity is of value and should always be looked for.

Myers<sup>15</sup> conclusions are of value: "In abscess of the cerebrum conjugate deviation of the head and eyes is extreme when the lesion affects the angular gyrus of the parietal lobe; it is of moderate extent if the lesion affects the posterior part of the frontal lobe close to the Rolandic area. It is absent, no matter how extensive the lesion, in abscess of the prefrontal region of the frontal lobe, in abscess



of the occipital lobe, and in abscess of the temporal lobe. The deviation in cerebral lesions, when present, is toward the side of the lesion. Interpreting the movement as an attitude of attention and assuming the angular gyrus to be the center of psychic vision (*e.g.*, the interpretation of visual stimuli), as shown by the fact that lesions of this region on the left side give rise to alexia or agnosia, the inability to understand writing or to recognize objects by vision, I conclude that attention is dominated most powerfully by stimuli arising within the sphere of vision and relatively little by other kinds of stimuli."

Anyone interested in this subject is referred to Meyers'<sup>15</sup> most excellent article.

Apparent agreement exists that conjugate deviation of the eyes is caused by supranuclear lesions; the fact that the internal rectus involved in conjugate deviation may function normally on convergence also places the lesion above the level of the nucleus.

Conjugate deviation of the eyes, especially when associated with evidence of cortical irritation, may be caused by irritation of the centers concerned and is probably, therefore, toward the side opposite the cerebral lesion. When associated with evidence of severe cortical damage, such as paralysis, it may be caused by paralysis of the centers involved and is probably, therefore, toward the side of the cerebral lesion.

It has value, when taken in conjunction with other findings, not only in lateralizing the lesion but also, in certain cases, in pointing to definite areas within the frontal lobe itself.

### C. INTRINSIC OCULAR MUSCLES

Involvement of the intrinsic musculature of the eye is revealed by the state of the pupil, which is the net result of the interplay of stimuli upon the sphincter and dilator mechanisms of the iris.

#### I. DILATATION

Active dilatation of the pupil (spastic mydriasis) is effected through the superior colliculus, the medial longitudinal bundle of the same side, the tectospinal fasciculus of the opposite side, Bridge's ciliospinal center in the intermediolateral cell column of the eighth cervical and first, second, and third upper thoracic segments of the same side, cervical trunk, superior cervical ganglion of the same side, and the internal carotid plexus. At this point the fibers divide; most of them go through the caroticotympanic nerve (or plexus), the cavernous and ophthalmic plexus, and both the long and short ciliary nerves to the dilator of the iris. A smaller number proceeds through the semilunar ganglion, the ophthalmic division of the fifth nerve, the nasociliary nerve, and the long ciliary nerves to the iris.

Fuchs<sup>8</sup> states that dilatation may be a spastic mydriasis often seen in states of cerebral irritation of the most varied sort or in stimulation of the cervical sympathetic, but that it is most commonly a paralytic phenomenon accompanying involvement of the oculomotor nerve and apt, therefore, to be found in association with paralysis of accommodation in ophthalmoplegia interna or with oculomotor paralysis in ophthalmoplegia externa.

Table 3 is the record of pupillary dilatation.

Bilateral dilatation of the pupils occurred twenty-six times; unilateral dilatation was reported seventeen times, ten on the same side, seven on the opposite side.

Of twenty-six cases of bilateral dilatation only one, a case without orbital disease, presented other evidence of oculomotor nerve involvement in the form of weakness of the levator palpebræ superioris of each side.

Light reaction was sluggish in twelve and absent in five.

A single lesion of the oculomotor nerves or the visual pathways themselves will not cause bilateral dilatation of the pupils, the finding present in approximately two-thirds of the cases in which the state of the pupil was recorded; any possible etiology must necessarily be so diffuse and general in its effect that the lower visual pathways and centers of each side are equally involved and paralytic mydriasis is equally present in each eye. Increased intracranial pressure, probably on the oculomotor nerves, is such a factor and is responsible for pupillary dilatation in frontal lobe abscess.

A consideration of these facts brings the realization that bilateral dilatation of the pupils means increased intracranial pressure and nothing more.

TABLE 3.—PUPILLARY DILATATION

Side	Number of cases	Oculomotor paralyses						Ipsilateral complete ext. ophthalmoplegia	Sluggish light reaction	Absent light reaction	Orbital disease
		Ipsilateral		Contra-lateral		Bilateral					
		Complete oculomotor paralysis	Ptosis	Fixed contracted pupil	Ptosis	Superior rectus paralysis	Ptosis				
Bilateral .....	26	..	..	..	..	..	1	..	12	5	..
Ipsilateral .....	10	1	4	7	..	1	..	1	2	2	1
Contralateral .....	7	..	..	..	1	..	..	..	1	1	..
Total .....	43	1	4	7	1	1	1	1	15	8	1

A study of Table 3 shows that lesions of extrinsic ocular muscles occurred more frequently in ipsilateral than in contralateral or bilateral dilatation; when diffuse, uniformly increased intracranial pressure, as indicated by bilateral dilatation, was present, such lesions were infrequent.

Unilateral dilatation is slightly more frequent on the side of the cerebral abscess than on the side opposite the abscess and has, therefore, some lateralizing value, which is increased by association with lesions of extrinsic ocular muscles.

## II. CONTRACTION

Pupillary contraction may be caused, not only by spasm of the sphincter (spastic miosis) but also by paralysis of the dilator (paralytic miosis) of the iris; most authorities assign the greater importance to variations in the tone of the sphincter.

Sphincter contraction is effected through the superior colliculus, the pupillary center in the anterior part of the Edinger-Westphal nucleus, and the oculomotor nerve; the superior colliculus receives stimuli from the ipsilateral optic tract (pupillary fibers from each retina), and from the ipsilateral visual cortex through the optic radiation.

Contraction may result, not only from light, but also from pressure on the visual pathways; Macewen<sup>13</sup> observed that the pupil is apt to be contracted when the cerebral abscess is small, dilated when it is large. Contraction also accompanies accommodation, a reflex which is integrated over an arc practically identical with that of light reaction; it also occurs in conjunction with convergence, but whether due to convergence or to the accompanying accommodation is a disputed point.

The sphincter of the iris is inhibited simultaneously with stimulation of the dilator through the cervical sympathetic by sensory stimuli from widely scattered skin areas, or by psychic stimuli from higher cerebral centers.

Fifteen observations on pupillary contraction were noted, one ipsilateral, two contralateral, and twelve bilateral; only three of these fifteen cases presented other evidence of oculomotor nerve involvement. Table 4 records the associated lesions in this group.

## SUMMARY OF PUPILLARY REACTIONS

Bilateral pupillary reactions are the rule because the stimuli of which they are the result are bilaterally effective in the lower visual centers.

Bilateral reactions are indicative of increased intracranial pressure only and have no localizing value.

Unilateral reaction, especially when accompanied by other evidence of oculomotor nerve involvement, has some value in indicating the laterality of the cerebral lesion.

Dilated and contracted pupils are frequently accompanied by sluggish or absent light reaction.

#### D. OPTIC NERVE

##### I. VISION

Observations regarding the vision were few in number and casual in character. The exact visual acuity was but rarely recorded; total blindness was occasionally noted.

TABLE 4.—PUPILLARY CONTRACTION

<i>Side</i>	<i>Number of cases</i>	<i>Oculomotor paralyses</i>				<i>Ipsilateral complete ext. ophthalmoplegia</i>	<i>Orbital disease</i>
		<i>Ipsilateral</i>			<i>Bilateral</i>		
		<i>Fixed dilated pupil</i>	<i>Internal rectus paralysis</i>	<i>Superior rectus paralysis</i>	<i>Superior rectus paralysis</i>		
Bilateral .....	12	..	1	1	..	..	..
Contralateral .....	2	2	..	..	1	1	1
Ipsilateral .....	1	..	..	..	..	..	..
Total .....	15	2	1	1	1	1	1

Five cases presented impaired vision in the ipsilateral eye, one in the contralateral eye, and fifteen in both eyes; orbital disease was more frequently present in unilateral than in bilateral impairment. The incidence of bilateral visual impairment was the same in the cases with and without orbital complication.

Foster Kennedy,<sup>11</sup> in speaking of the effect on vision of a frontal lobe tumor (or abscess), makes the statement that when such a lesion is situated in the central part of the frontal lobe direct pressure on the ipsilateral optic nerve is less apt to be present than is general increase of pressure, which causes bilateral papilledema without visual impairment. When the lesion is situated in the basal part of the frontal lobe, direct downward pressure on the optic nerve may cause ipsilateral compression neuritis and impaired vision, followed by



primary optic atrophy; accompanying cerebral edema may cause papilledema with good vision in the opposite eye (basal frontal syndrome).

## II. FUNDUS

Unilateral fundus lesions, summarized in Table 5, occurred most frequently on the side of the cerebral abscess.

Bilateral fundus findings, without record of visual acuity, are recorded in Table 6; the greater involvement occurred most frequently on the side of the cerebral lesion.

Evidence of stasis, as manifested by congestion of retinal veins or by choked disc (papilledema) was present in 84 percent of the cases in which fundus notes were recorded; choked disc was present in 46 percent (16 percent of the entire group of 204 cases).

TABLE 5.—UNILATERAL FUNDUS LESIONS

Lesion	Side	
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>
Congestion .....	3	1
Papilledema .....	2	..
Choked disc .....	1	..
Optic neuritis .....	5	1
Atrophy .....	1	..
Total .....	12	2

TABLE 6.—BILATERAL FUNDUS LESIONS

Lesion	Number of cases	Most marked on	
		<i>Ipsi-lateral</i>	<i>Contra-lateral</i>
Congestion ....	14	..	..
Papilledema ...	11	3	1
Choked disc ...	36	6	1
Optic neuritis..	13	1	2
Atrophy .....	4	1	..
Total .....	78	11	4

This small material indicates that fundus disease in abscess of the frontal lobe is due primarily to disturbance of circulation rather than to inflammation

It is not within the province of this paper to discuss at length the mechanism of fundus lesions or to attempt an evaluation of the factors in their production, inflammation, toxic or chemical irritants, or stasis within the sheath of the optic nerve, due to direct downward pressure or to interference with the circulation of the cerebrospinal fluid. The opinion is now quite generally held that papilledema and choked disc are synonymous terms, and that optic neuritis introduces an inflammatory reaction, from whatever cause. Confusion may have been present, especially in the minds of the older authors, with regard to the exact differentiation between optic neuritis and choked disc; Fuchs<sup>8</sup> remarks that the distinction is better established theoretically than in practice.

Benedict<sup>4</sup> states that the consensus of opinion is that frontal lobe abscess causes choked disc rather infrequently and has but little localizing value for the reason that the cerebral edema responsible for the disc changes is not always constant, or present only on the side of the abscess.

Lillie,<sup>14</sup> in a series of eleven cases of encapsulated frontal lobe abscess, observed bilateral choked discs, varying from one to six diopters, in eight cases; vision was good and fields revealed only a mild concentric contraction for form and color. Halstead and Vaughan<sup>10</sup> found optic neuritis and choked disc frequently present and greater on the side of the lesion, due to direct pressure on the nerve.

Coleman<sup>5</sup> noted no relation between the size of the abscess and the degree of choking, inasmuch as the latter may be due more to encephalitis or massive cerebral edema than to the abscess.

### III. VISUAL FIELDS

Reports of careful visual field examinations were rarely encountered; fields normal on gross testing were but infrequently noted. It may, therefore, be of interest to refer to Eagleton's writings, which emphasize the value of field studies in the diagnosis of intracranial lesions.

Eagleton,<sup>7</sup> in his monograph on brain abscess, published in 1922, and in later writings,<sup>6</sup> dwells on the importance of cerebral edema in the production of disc and field changes. It is a factor constantly present in intradural suppurative lesions and varies much in extent and degree; the greatest edema is not necessarily on the side of the lesion. Edema may manifest its presence by uneven, transient, hemianoptic indentation of the contralateral field, the variability of the edema is reflected in changing fields which necessitate frequent examinations, the value of which is greatest when the diagnosis is as yet uncertain.

When the cerebral edema is generalized, as is the case when it has extended from the posterior fossa to the great brain, the fields usually show slight general contraction with hemianoptic tendency.

Lillie observes that variations in cerebral edema, as indicated by changing discs and fields, point to an intradural process not yet stabilized (unencapsulated abscess); surgery is more apt to be satisfactory when encapsulation, indicated by stable discs and fields, has been established.

Lillie,<sup>14</sup> Benedict,<sup>4</sup> and others point out that symptoms of encapsulated abscess are those of tumor similarly situated and that abscess presenting tumor symptoms is most amenable to surgery.

## E. PYRAMIDAL TRACT

Pyramidal tract involvement, frequent in this study, was manifested by muscular twitching, general or local (Jacksonian attacks), or by weakness of muscle groups or body halves (paresis, paralysis, hemiparesis or hemiplegia).

The proximity of the cortical motor centers, the internal capsule, and the corticospinal and corticobulbar motor tracts to the posterior portions of the frontal lobe offers a ready explanation of these symptoms; the fact that frontal lobe lesions exert their effects on these pathways before decussation explains the development of most pyramidal tract symptoms on the contralateral side of the body. Such symptoms on the ipsilateral side, more frequent in lesions of the posterior fossa, are due to dislocation of the medulla against the basilar plate of the occipital bone with resultant pressure on the pyramid of the opposite side above decussation, or to herniation of the medulla into the foramen magnum with pressure on the ipsilateral pyramidal tract below decussation, in either case with resultant ipsilateral symptoms. Such symptoms occur but rarely in supratentorial lesions; they may be caused by shifting of the brain with resultant pressure on the contralateral pyramid by the tentorium cerebelli. Myers<sup>16</sup> suggests that physiological or anatomical variations in the pyramidal tracts, such as preponderance of ipsilateral fibers, or even absence of decussation, may be responsible for such findings.

Consideration of these facts precludes the possibility of attaching absolute lateralizing value to such signs; they may be highly suggestive but must be evaluated in connection with other factors present.

Generalized twitching, present in two cases, is indicative of widespread cortical irritation caused by increased intracranial pressure.

Ipsilateral localized twitching, significant of irritation of the opposite motor cortex, appeared more frequently than did contralateral twitching, indicative of cortical irritation on the side of the lesion.

Bilateral twitching was frequently present; it has no lateralizing or localizing value.

The incidence of unilateral and bilateral localized twitching is recorded in Table 7.

Generalized convulsions, indicative of increased intracranial pressure, were present in fifty-nine cases, with loss of consciousness in seventeen. This finding has, of course, no localizing value.

Table 8 summarizes localizing symptoms which appeared in connection with general convulsions; such symptoms were more frequent

TABLE 7.—LOCALIZED TWITCHING

Site	Side		
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>	<i>Bi-lateral</i>
Face .....	2	1	3
Arm .....	4	2	3
Hand .....	..	1	1
Leg .....	3	..	2
Total .....	9	4	9

TABLE 8.—GENERALIZED CONVULSIONS

Beginning in:	Side	
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>
Face .....	1	2
Arm .....	..	1
Hand .....	..	1
Total .....	1	4
More marked on one side of body .....	1	4

on the contralateral side of the body, indicating cortical irritation on the side of the lesion. Ballance<sup>2</sup> makes the statement that the initial local spasm of a fit or a local paresis is pathognomonic of the site of the lesion.

The etiology and localizing value of Jacksonian attacks does not need statement here; a study of Table 9 shows that, while contralateral extremities were involved more frequently than those of the ipsilateral side, the two sides of the face were equally involved. The tendency of disturbances of facial musculature to appear with equal frequency on the two sides has been noted elsewhere.

Th occurrence of contralateral and bilateral Jacksonian attacks in cases of solitary cerebral abscess necessitates caution in the interpretation of unilateral manifestations.

Table 10 illustrates very well the decided contralaterality of lesions characterized by impairment or loss of muscular function; the incidence of such lesions on the two sides of the face is much more nearly the same than that of lesions occurring elsewhere.

The occurrence of paresis or paralysis on either side of the body lessens the lateralizing value of all unilateral findings.

#### SUMMARY OF PYRAMIDAL TRACT SYMPTOMS

The significance of symptoms dependent upon involvement of the motor cortex and tracts may be summarized by saying that general convulsions and generalized twitching have no lateralizing or localizing value; they mean only increased intracranial pressure. Unilateral Jacksonian attacks have definite lateralizing and localizing value, pointing directly to the precentral gyrus; when bilateral they



TABLE 9.—JACKSONIAN ATTACKS

Site given	Side		
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>	<i>Bi-lateral</i>
Face .....	2	2	1
Arm .....	3	7	4
Leg .....	1	3	5
Total .....	6	12	10
Site not given..	1	10	..

TABLE 10.

## A. PARESIS AND PARALYSIS

Site	Side	
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>
Face .....	15	19
Arm .....	2	34
Hand .....	1	8
Leg .....	1	20
Total .....	19	81

## B. HEMIPARESIS AND HEMIPLEGIA

Side	
<i>Ipsilateral</i>	<i>Contralateral</i>
3	31

mean only increased pressure. Partial or complete loss of muscular power in one member or one-half of the body has both lateralizing and localizing value; when the muscular system of half the body is involved an extensive lesion of the precentral gyrus or a localized lesion in the internal capsule or motor tracts is indicated.

An attempt was made to associate the site of the lesion with the symptoms present; this was impossible in most cases owing to the lack of precise information concerning the exact location of the abscess; such information was occasionally given, but in the majority of cases the findings at operation or autopsy were not reported accurately enough to be so used.

## F. MOTOR CRANIAL NERVES NOT ELSEWHERE DISCUSSED

The nuclei of the cranial nerves have a bilateral crossed cortical connection which is effected through the pyramidal tracts, principally that of the opposite side; the variability of these pathways, commented on in discussing their lesions, is reflected in the variable cortical representation of cranial nerves.

Irritative symptoms, due to stimulation of the motor cortex, appear on the opposite side of the body; paralytic lesions, if central (above the decussation of the pyramids) also appear on the opposite side of the body, as do the symptoms of pyramidal tract involvement with which they are frequently associated; paralytic lesions, caused by direct or transmitted pressure on the peripheral nerve, appear on the side of the pressure. Unilateral paralysis as the result of an

upper motor neuron (central) lesion is rendered unlikely by the bilateral cortical connection of the cranial nuclei; most such lesions are peripheral.

TABLE 11.—MOTOR CRANIAL NERVES NOT HITHERTO CONSIDERED

Nerve	Paralysis			Twitching			Spasm			Jacksonian attacks		
	<i>Ipsilateral</i>	<i>Contralateral</i>	<i>Bilateral</i>	<i>Ipsilateral</i>	<i>Contralateral</i>	<i>Bilateral</i>	<i>Ipsilateral</i>	<i>Contralateral</i>	<i>Bilateral</i>	<i>Ipsilateral</i>	<i>Contralateral</i>	<i>Bilateral</i>
Trigeminal (mandibular branch) .....	..	1*	..	..	..	..	..	..	..	..	..	..
Facial .....	15	19	..	2	2	6	..	..	..	4	4	1
Vagus .....	..	..	{4†}	..	..	..	..	..	1‡	..	..	..
Accessory .....	..	..		..	..	..	..	..	..	..	..	..
Hypoglossal ...	2§	4§	..	..	..	..	..	..	..	..	..	..
Total .....	17	24	4	2	2	6	..	..	1	4	4	1

\* Paralysis of pterygoid muscle with jaw deviation. † Paralysis of pharyngeal constrictors.  
‡ Laryngeal spasm. § Paralysis of tongue.

A glance at Table 11 shows that the facial nerve was most frequently involved. Irritative symptoms appeared with equal frequency on the two sides of the face; paralytic lesions were slightly more frequent on the contralateral side. This was true, not only of the facial nerve, but also of the motor division of the trigeminal and of the hypoglossal.

Bilateral symptoms, such as pharyngeal paresis and laryngeal spasm, were undoubtedly caused by pressure on bulbar nuclei.

Pressure of the cerebral abscess itself may cause peripheral paralytic lesions on the ipsilateral side; such lesions on the contralateral side can be explained only on the basis of pressure from cerebral edema.

Variations in the pressure of cerebral edema offer a reasonable explanation for the more frequent occurrence of paralytic lesions of the trigeminal, facial, and hypoglossal nerves on the contralateral instead of the ipsilateral side, as was the case in lesions of the oculomotor, trochlear, and abducens nerves. Such variations but emphasize the fact that any lateralizing significance possessed by unilateral lesions of cranial nerves is but limited.

## G. SENSORY DISTURBANCES

The literature on disturbance of sensation in frontal lobe abscess is scant and but few notes concerning it were found in the case reports examined.

Cortical sensory representation is, like cortical motor representation, largely crossed; afferent sensory tracts, whether those of pain, temperature, light touch, or discriminative touch, decussate almost completely, if not entirely, before leaving the upper level of the medulla. Symptoms caused by a lesion above the medulla, such as abscess of the frontal lobe, are on the opposite side of the body. It is only reasonable to assume that here, as in the case of ipsilateral pyramidal tract signs, direct fibers may occasionally be present in unusually large numbers and so account for ipsilateral sensory disturbances.

The mechanism responsible for sensory disturbances is that involved in the production of motor symptoms, *vis.*, transmitted or direct pressure, or actual involvement of the cortical centers or fiber tracts entering or leaving the internal capsule; the location of the cortical sensory area in the postcentral gyrus lessens the incidence of sensory as compared with motor symptoms.

Ballance<sup>2</sup> observes, in discussing frontal lobe tumor, that there is no loss of common sensation on the opposite side of the body when the sensory cortex is involved, but that there is loss of muscle sense and of the power of localizing light touch.

Osnato<sup>19</sup> mentions as symptoms due to involvement of the sensory cortex disabilities connected with the recognition of size, form and shape of objects, and disturbances in deep muscle sensibility and in discrimination between one and two points which are ordinarily recognized at 3 mm. or less; in frontal lobe abscess such points may not be recognized at much wider spacing.

Anesthesia of the ipsilateral face was present in one case, hyperesthesia of the contralateral face and cornea in one case. Bilateral corneal anesthesia was noted once. Impaired sensation of the opposite side of the body was found twice, and numbness of the ipsilateral side once. Disturbed sensation was present once in each arm.

Anosmia, due to direct downward pressure on the olfactory nerve, is a not uncommon symptom in both frontal lobe tumor and abscess. In this series of cases it was noted twice. It doubtless would be found more frequently by careful examination.

This material, too scanty to be of value, suggests the advisability of looking for disturbances of sensation in frontal lobe abscess cases whenever possible.

## H. REFLEXES

The notes on reflexes in this series of cases were disappointing because of brevity and incompleteness. In many cases there was only a casual observation on one reflex, giving the impression of a hurried and incomplete neurological examination. In a few cases a complete examination had been made and the findings recorded; in still fewer instances were there progress notes on the variations in the reflexes.

Reflex mechanism is complicated, depending on spinal cord pathways, which are, in many cases, known in detail, and on extensive ramifications, both in cord and cortex, which are not so well known. The study of reflexes may be expected to yield information concerning the integrity of the spinal reflex arc, its connections with the cortex, and the control which the cortex exercises upon the lower centers.

Superficial reflexes are phylogenetically younger than deep reflexes and are dependent on normal cortical activity. Increased cortical activity or tone in the neuromuscular mechanism increases superficial reflexes; lowered activity or tone may cause their disappearance. Any lesion which breaks the spinal arc in the anterior horn cell, efferent motor nerve, afferent sensory nerve, or in the posterior root, will obviously ablate these reflexes, as will a lesion of the upper motor neuron, presumably by removal of the activating action of the cortex. The pathways through the cortex are not as well understood as are the spinal arcs; the pyramidal fibers pursue a well-known course, but there are probably tracts from the premotor area with important connections, whose course is not well known.

The deep reflexes are phylogenetically older than the superficial and are controlled in part by the inhibitory action of the cortex. Lesions of the upper motor neuron remove this inhibitory action and cause hyperactive deep reflexes; lesions of the spinal arc or posterior columns may destroy them entirely. Numerous deep reflexes, such as the ankle clonus or Babinski, are in reality the manifestations of increased neuromuscular tone due to removal of cortical inhibition, or to meningeal or posterior root irritation.

Superficial and deep reflexes are profoundly affected by the state of mind and body and reflect accurately conditions such as increased mental activity or shock; under such circumstances reflexes have no localizing value. When discrepancies appear between the superficial and deep reflexes, or between reflexes of opposite sides or of different levels, or when abnormal reactions are persistently present, the finding assumes value.



Abscess of the frontal lobe affects superficial and deep reflexes by disturbing their cortical associations through generalized pressure or actual destruction of nerve tissue.

Reflex changes noted in this study were almost entirely bilateral; only two were unilaterally present in numbers sufficient to give them any importance; there were many isolated findings unrelated to anything else of diagnostic importance; manifestly they were of no value.

Table 12 records the notes on superficial reflexes.

TABLE 12.—SUPERFICIAL REFLEXES

<i>Abdominal Reflex</i>	<i>Number of cases</i>	<i>Accompanying Reflexes</i>						
		<i>Hyperactive cremasterics</i>	<i>Kernig's sign present</i>	<i>Achilles' jerk present</i>	<i>Babinski's sign present</i>	<i>Ankle clonus present</i>	<i>Knee-jerks hyperactive</i>	<i>Knee-jerks absent</i>
Hyperactive:								
Contralateral .....	1	1	1	1	..	..	..	1
Bilateral .....	3	..	..	..	..	..	..	..
Absent:								
Ipsilateral .....	1	..	3	2	2	2	1	3
Contralateral .....	1							
Bilateral .....	8							
Total .....	14	1	4	3	2	2	1	4

Hyperactive superficial reflexes are due to increased cortical activity or irritability of the neuromuscular system; this was borne out, in this small group, by the hyperactive cremasterics and the positive Kernig, the latter indicative of meningeal irritation, especially about the posterior roots.

The abdominal reflexes were bilaterally absent in eight cases and unilaterally absent once on each side; the accompanying reflexes were indicative, in almost every case, of upper motor neuron impairment. Babinski's sign, the Achilles jerk, and the ankle clonus were each present twice in this small group; Kernig's sign was positive three times; the knee-jerks were hyperactive once and absent three times. These findings suggest a serious lesion, either meningeal or of the upper motor neuron.

Changes in the deep reflexes, summarized in Table 13, were more commonly noted. Bilateral Achilles jerk was present four times; the accompanying deep reflexes indicated either meningeal irritation

TABLE 13.—BILATERAL DEEP REFLEXES

Achilles' jerk .....	4
Ankle clonus .....	8
Babinski's sign .....	7
Knee-jerks hyperactive .....	10
Knee-jerks absent .....	8
Kernig's sign .....	24

TABLE 14.—UNILATERAL DEEP REFLEXES

Reflex	Side	
	<i>Ipsi-lateral</i>	<i>Contra-lateral</i>
Ankle clonus .....	2	7
Babinski's sign .....	2	5

or upper motor neuron disturbance. Ankle clonus was present eight times, Babinski's sign, seven times, hyperactive knee-jerks, ten times; knee-jerks were absent eight times. These findings were bilateral and associated with various combinations of deep reflexes which were without particular pattern.

Kernig's sign was present in twenty-four cases, being the reflex most frequently reported.

Unilateral deep reflexes (Table 14) were reported infrequently; crossed motor and sensory innervation explains their more frequent occurrence on the contralateral side.

Spasticity was reported once in each arm and once in both arms; it was present once in the ipsilateral leg and twice in both legs.

Loss of control of bowel and bladder sphincters was noted in ten cases; in five, loss of bladder control was present without bowel disturbance. These findings have the same general significance as that attributed to the presence of the deep reflexes noted above.

#### SUMMARY OF REFLEX CHANGES

The bilateral reflex changes noted in this group of frontal lobe abscesses were without lateralizing or localizing value; they indicated meningeal irritation or ablation of upper motor and sensory neuron function. Unilateral changes occurred most frequently on the side opposite the lesion.

#### EQUILIBRIUM AND COORDINATION

Disturbances in equilibrium and coordination were but rarely noted, either in the case histories or literature studied. Staggering gait before operation was noted in one case; in one an ataxia, otherwise undescribed, was present on both sides of the body. In one case an ataxia was present only on the side contralateral to the lesion.

Nystagmus directed to both sides was present in four cases, to the contralateral side only, in one case. Adiadokokinesis was present on the contralateral side in one case. These findings comprise the entire list of such disturbances in this series of cases.

Frontal ataxia has been described by various writers. Ballance<sup>2</sup> states that in this condition the patient sways to either side when standing and would fall if not supported; in walking he does not have the zig-zag staggering gait of cerebellar lesions. The ataxia is attributed to lack of attention or possibly paresis of the muscles of trunk and neck. Osnato<sup>19</sup> and Wechsler<sup>23</sup> attribute this symptom to disturbance of the fronto-ponto-cerebellar and fronto-parietal-occipital tracts connecting the frontal lobe with the opposite cerebellar hemisphere. In man there is a large tract from the frontal cortex directly to the pons; from here fibers carry the impulses to the opposite cerebellar hemisphere via the middle cerebellar peduncles. This is so well developed that it is surprising that symptoms are not more frequent in extensive frontal lesions.

Foster Kennedy<sup>11</sup> makes the statement that the only posture characteristic of such disease (frontal lobe tumor) is titubation on the heels and a tendency to retropulsion. Such disturbances are important only in that they may simulate a cerebellar lesion.

Nystagmus was but rarely mentioned; it was present in five cases, in four of which it was directed to both sides. Grinker<sup>9</sup> states that the communication between the cranial cavity and internal ear permits the transmission of variations of intracranial pressure to the latter organ, a very possible cause for the symptom. It may also be due to pressure on the vestibular nerve, tracts, or nuclei.

### I. APHASIA

Twenty-nine cases presented speech difficulties. In three the condition was termed aphasia, in one, sensory aphasia without agraphia, and in one, paraphasia with word blindness; in the others the speech was described as impaired, slurred, slow, or lost.

Aphasia is described by Wechsler as "a disturbance in the highest speech mechanism, in the realm of speech concepts, in the sphere of symbolic thinking. In aphasia the formulation of propositions and their relations is impaired or lost. The disturbance is in the comprehension, elaboration, and expression of speech concepts." Older views held that speech was a higher faculty capable of dissociation by disease into lesser faculties, each located in a more or less definite anatomical center, destruction of which caused the so-called pure aphasias, such as visual aphasia or auditory aphasia, sensory or motor agraphia.

Recent work, notably that of Head (quoted by Wechsler<sup>23</sup>), indicates that the mechanism of aphasia is not as simple as was at first believed. The newer concept of aphasia, as defined by Wechsler,

involves the idea that speech is a highly intellectual process, the "symbolic thinking" of Head, which breaks down, not into sub-faculties, or pure aphasias, but into broader, more inclusive concepts of speech comprehension and expression, not located in definite anatomical centers. Aphasia in any of its forms affects the whole intellectual process, even though a particular concept is especially involved.

On the basis of this theory several types of aphasia are recognized. Verbal defects, characterized by defective power of forming words, either for internal or external use, with intact comprehension, correspond to the motor or expressive aphasia of the older terminology. Syntactic defects are disorders in the balance and rhythm of symbolic expression, comparable to word deafness. Nominal defects are difficulties in the designation of objects by name (global aphasia); the defects in writing and reading here involved bear the older terminology of agraphia and alexia. Semantic defects involve difficulties in the comprehension of words and phrases as a whole (Head, quoted by Grinker<sup>9</sup>).

While the theory of definite centers for so-called pure aphasias is no longer universally accepted, clinical experience teaches that lesions in certain areas produce certain types of speech disturbance, which may, therefore, have localizing value. It is thought that the speech zone is in the cerebral hemisphere opposite the dominant hand. According to Wechsler,<sup>23</sup> left frontal lesions situated low in the pre-Rolandic area disturb spontaneous speech and expression of language; when such defects are associated with Jacksonian epilepsy or muscular disturbance of the opposite side the pre-Rolandic area is still more definitely indicated as the site of the lesion. When comprehension of speech is mainly involved, the lesion is generally behind the Rolandic and below the Sylvian fissure, the upper part of the temporal lobe and the angular gyrus.

In this group of cases speech difficulty was observed in twenty-nine, but the designation impaired, slurred, slowed or lost, does not fully describe the difficulty; it must be remembered that very sick patients frequently have speech difficulties, due to weakness and mental confusion, that are not true aphasias. Lesions of the frontal lobe are early characterized by inattention and lack of interest; drowsiness and apathy, attendant on the development of increased intracranial pressure, further complicate the mental picture (Grinker<sup>9</sup>). These factors may so interfere with speech as to simulate an aphasia which cannot be ruled out without comprehensive neurological examination.



The reduction of increased intracranial pressure by the injection of hypertonic salt solution or by ventricular drainage may for a time relieve the mental symptoms due to pressure and allow the disturbances caused by the lesion itself to stand out more clearly.

In these twenty-nine cases of speech disturbance the abscess was in the left lobe in fifteen cases and in the right in fourteen; approximately two-thirds of each group gave evidence, in the form of Jacksonian attacks, paresis or paralysis, of involvement of the ipsilateral motor cortex. Only two of the fourteen right-sided abscesses were found in the motor area; of the fifteen left-sided abscesses seven were located in the motor area (46 percent as contrasted with 14 percent in right-sided lesions).

It is more than probable that some of the speech difficulties observed in both right- and left-sided lesions were not true aphasias, but abnormalities of expression due to other conditions, such as shock or toxemia.

The most exact localizing information derived from the presence of aphasia is found in cases in which it is associated with clear-cut Jacksonian attacks on the side of the dominant hand; under such circumstances the lower portion of the precentral gyrus on the side opposite the dominant hand is almost certainly involved.

## J. PSYCHIC CHANGES

The frontal lobes, particularly the left, are thought to be the seat of the higher psychic functions of the brain and their lesions are commonly accompanied by disturbances in the mental and psychic life of the patient. Ballance<sup>3</sup> notes, in subacute or chronic frontal lobe abscess, evidence of psychic dissolution with loss of the highest functions of the brain, such as ideation, memory, control, attention, and judgment. Foster Kennedy,<sup>11</sup> speaking of frontal tumors, considers mental changes one of the most reliable guides; he enumerates lessened power of attention, irrelevant replies, trivial and meaningless jocosity (*Witzelsucht*), inept and causeless laughter, hypersensitiveness to offense, effusive apologies for trifles, *petit mal* with sudden attacks of mental confusion, and *hebetude*, from which the patient is aroused with difficulty and which later passes into stupor.

Osnato<sup>19</sup> remarks on the presence of euphoric states, characterized by flippant and discordant emotional reaction in the direction of well being, a reaction out of proportion to the seriousness of the illness. Carelessness in dress and personal habits and inattention to and loss of interest in their business, home, or even their own illness, may not infrequently be seen in patients with frontal lobe abscess.

Changes in personality are common; a patient naturally polite, co-operative, and obedient becomes rude, obstreperous, and unruly. Obscenity and profanity may be indulged in.

Most of these mental and psychic changes were present in this series of cases. Mental sluggishness was the most common; it was noted in twenty-one cases. Loss of memory was present in fourteen and personality changes in twenty. In one of my own cases the only thing indicative of disturbed intelligence was the momentary inability to remember his own name when entering the hospital; it did not appear again during the entire illness.

Such mental and psychic disturbances, while suggestive, possess no localizing value.

### SUMMARY

The results of an analysis of neurological symptoms noted in 204 cases of solitary abscess of the frontal lobe may be summarized as follows:

A. Neurological symptoms of frontal lobe abscess may be divided into two groups. In one group, composed of symptoms indicative only of increased intracranial pressure and, therefore, without localizing value, are bilateral and generalized manifestations; in the other group are symptoms caused by involvement of centers and tracts in or near the frontal lobe, neighborhood symptoms which have, therefore, some lateralizing and localizing value. Most unilateral and localized manifestations belong in this group.

In the first group are included the following ocular lesions, all bilateral: paralyzes of extrinsic muscles, pupillary changes, visual impairment, and the retinal, disc, and field changes.

Also included in this group are bilateral and generalized twitchings, convulsions, and paralyzes.

Most reflexes, both superficial and deep, belong in this group; psychic and mental changes are also included.

In the second group, composed of symptoms which may have some lateralizing or localizing value, are unilateral lesions of extrinsic ocular muscles, conjugate deviation of the eyes, unilateral pupillary changes, unilateral disturbances in vision, retina, optic nerve, and visual field.

Unilateral or localized twitching, spasm, or paresis is of value; Jacksonian attacks, especially when associated with aphasia, may be of definite localizing value, as may hemiparesis and hemiplegia.

Unilateral reflexes, especially when persistently or repeatedly present, are of value in suggesting the side of the lesion.

Aphasia, especially when associated with Jacksonian attacks, has definite localizing value.

B. Symptoms of frontal lobe abscess may be ipsilateral, contralateral, or bilateral.

Unilateral symptoms occurred more frequently on the side of the cerebral abscess than on the side opposite the abscess; they are, therefore, of some lateralizing value.

The frequent occurrence of unilateral symptoms on the side opposite the cerebral abscess limits their lateralizing and localizing value and necessitates caution in their interpretation.

Bilateral or generalized symptoms are indicative only of increased intracranial pressure.

### CONCLUSIONS

1. There is no neurological picture which is pathognomonic of frontal lobe abscess.

2. Neurological signs occur in frontal lobe abscess with sufficient frequency and possess sufficient localizing value to be of definite, and at times considerable assistance, not only in making the diagnosis but also in localizing the lesion.

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# TRACHEOHYPHARYNGEAL FISTULIZATION\*

(A NEW PROCEDURE FOR SPEECH PRODUCTION IN THE LARYNGECTOMIZED PATIENT)

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The plight of the laryngectomized patient is indeed an unenviable one. In spite of his successful recovery from a most formidable operative procedure and his probable rescue from certain death, his mental status is anything but a happy one. His lack of speech is a decided handicap. Economically it frequently prevents his return to his former vocation, thus establishing him in the status of a financial burden on his family, friends, or the community. Psychologically his unhappiness is predicated not only on his inability to work for his daily bread, but also upon the fact that his waking hours are unoccupied and there is nothing to distract his mind from continuous introspective brooding upon his unfortunate status. His lack of speech makes for difficult social intercourse and he instinctively rebels against what he pictures as an exhibition of pity for his position. These factors are obviously the mechanics in the production of an unhappy introverted existence.

Another phase of the aphonic state of the laryngectomized patient is also of sufficient import to warrant study. It is the frequent decision on the part of the patient not to submit to the operation of laryngectomy because of the inevitable state of speechlessness that follows. Not a few patients have refused this life-saving procedure on that account and have, therefore, progressed to the certainty of death. The fear of being transformed into a mute person is an important factor in the patient's outright refusal of laryngectomy or in his hesitation while valuable time is being lost.

It is obvious that the social, and often the economic rehabilitation of the laryngectomized patient will depend to a great extent on our ability to effect some means of vocalization in these unfortunate individuals. In the past our efforts have been mainly along the lines of the establishment of buccoesophageal speech or in the use of an artificial larynx.

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\* Candidate's thesis.

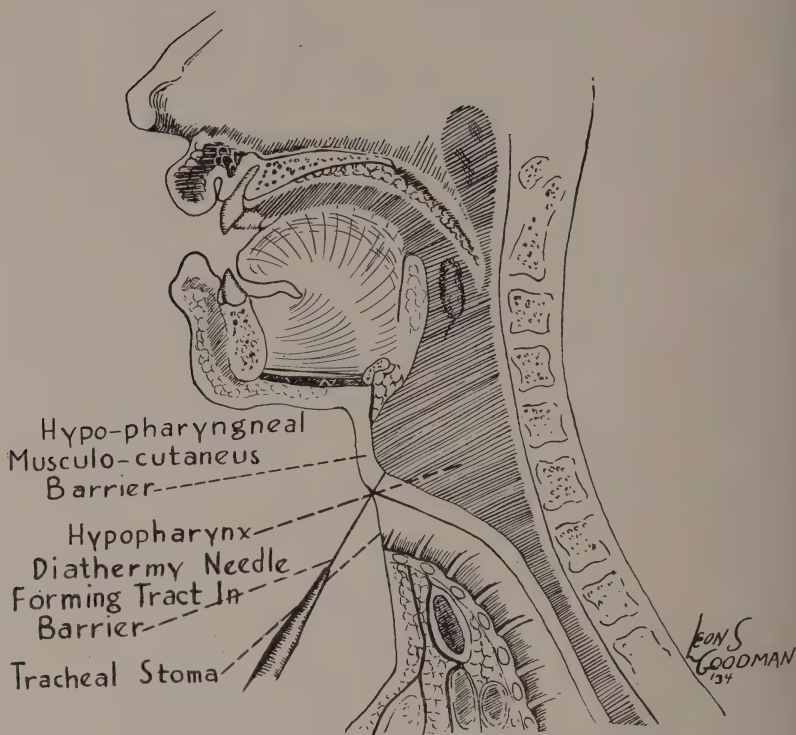


FIG. 1. Operation of tracheohypopharyngeal fistulation.

The speech apparatus in man consists of a mechanism for tone production—the vibrating vocal cords within the larynx, a mechanism for affecting a change in pitch—the intra- and extra-laryngeal musculature, and a mechanism for tone modulation in the production of speech—the tongue, palate, teeth and lips. The anatomical structures that make up the tone producing and pitch changing mechanisms are removed in the operation of laryngectomy, leaving those structures that make for modulation and speech more or less intact. It follows that the laryngectomized mute must have a means of tone production and change in pitch effected for him in order that he may speak once more. A fair amount of success has been attained in producing a mechanism to effect tone production, but pitch alteration as yet has not been consistently successful. The two methods of tone production are the buccoesophageal voice and an artificial larynx.

The development of speech methods in laryngectomized individuals paralleled and more or less coincided with the evolution and perfection of laryngectomy as a means of treating carcinoma of the

larynx. In 1859 Czermak,<sup>1</sup> followed by Bruecke somewhat later, constructed one of the earliest artificial larynges. The apparatus was attached to the tracheal canula at one end and had a tube leading to the oral cavity at the other end. While some sound was produced, the apparatus was more or less ineffectual. Bilothe, who performed the first total extirpation of the larynx in 1873 also had created with Gussenbauer<sup>6</sup> an artificial larynx, consisting of a voice box for tone production which was attached by a tube to the trachea and also to the pharynx by another tube that passed through a hypopharyngeal opening into the pharynx. Tone production was more satisfactory with this apparatus. The Braun<sup>2</sup> instrument, which also utilizes a hypopharyngeal fistula was produced later. Bruns<sup>3</sup> in 1861 improved upon the mechanism by substituting flexible tubing for the stiff metal ones of the original apparatus and, what is fundamentally more important by using rubber for the vibrating band. This produced a marked improvement in the quality of speech effected. Stoerk<sup>4</sup> devised a whistle in 1887 which was actuated by the bechic blast from the trachea. The whistle was held between the teeth and tongue and the subsequent tone was modulated into speech, which, however, was not altogether satisfactory. He made an additional change in his instrument by utilizing a bellows for the production of a continuous flow of air instead of the intermittent blasts from the trachea. Secretions that entered the tone producing mechanism either from the pharynx or trachea would cause frequent failure of a function and Wolf modified the mechanism so as to afford protection against the entrance of secretions. Gottstein<sup>5</sup> later suggested the combined Czermak-Stoerk apparatus—namely, the Czermak tone producing mechanism with flexible tubing introduced into the mouth alongside the buccal aspect of the teeth. Hochnegg produced an apparatus that utilized a bellows and a nasal tube that introduced the vibrating column of air into the nasopharynx.

Gluck<sup>7</sup> and Sorenson besides popularizing the operation of laryngectomy spent a great deal of time and effort in the construction of a variety of artificial larynges, utilizing decorative cabinets, arm and electric powered bellows and finally, a phonographic record for tone production—the tone then being led into the nasopharynx and then modulated into speech. Fundamental changes have been few since that period excepting Onodi's<sup>8</sup> use of a microphone to carry the sound into the nasopharynx. MacKenty,<sup>9</sup> Tapia,<sup>10</sup> Leyro-Diaz,<sup>11</sup> Casasdesus,<sup>12</sup> Hinoyar,<sup>13</sup> McKesson and Sheard have produced the modern types of artificial larynges.

They utilize a rubber reed or thin sheet metal vibrating mechanism for tone production, energized by a blast of air from the trachea or a bellows and utilizing a tube to convey the tone into the oropharynx. These later instruments are very efficient and quite satisfactory from the point of view of speech production. Unfortunately, many patients do not take kindly to any sort of a prosthesis, and we have had several patients refuse to use them for this reason alone. In others the apparatus causes some irritation and cough which may lead to its discontinuance. The monotony of the tone produced may be objectionable to some. Another factor that was of some importance in the past was the matter of cost. However, the new Sheard instrument can now be obtained for about \$20 which is very inexpensive. Most of these objections are of no great importance, excepting the patient's reluctance to employ an artificial prosthesis and his embarrassment from the attention that the instrument attracts. The only real objection to the modern artificial larynx is the fact that the pitch cannot be flexibly varied so as to produce inflections in speech, and that some training is necessary in order to obtain good results. The difficulty is in the proper actuation of the vibrating reed. Training is necessary to correctly perform this, just as the precise actuation of a reed in a musical instrument requires practice. The writer has attempted to overcome this latter objection by using an electrical vibrator similar to a buzzer. The tone produced, however, is not sufficiently intense. At the present time some work is being carried on in the employment of an oscillating vacuum tube with the production of a tone. The difficulty is in obtaining a small portable hidden or invisible mechanism.

*Pseudo-Voice.*—The second method of producing speech in the laryngectomized mute is by means of the so-called pseudo-voice. The voice is produced by swallowing air into a natural reservoir such as the stomach, esophagus or hypopharynx and then propulsing the air forward through some narrowing in the oropharyngoesophageal tract. The passage of the air by the constricted portion of the tract produces a tone that can be modulated by the teeth, tongue, palate, and lips into audible speech. Czermak,<sup>1</sup> described the first recorded pseudo-speech in 1859 in a case of complete laryngeal stenosis. The first published case of pseudo-voice in a laryngectomized patient was that of Stoerk<sup>4</sup> in 1887. Seiler<sup>15</sup> reported a case in 1888 and in the following year Strubing and Landois<sup>14</sup> published their investigation of Schmid's case. Woods reports that Salis-Cohen demonstrated a case in Philadelphia in 1895. In 1900 Gottstein<sup>5</sup> reviewed his results in a case operated upon by Mikulicz. He was able to teach his patient not only to talk



but also to sing with a range of about one octave. As with the development of the laryngeal prothesis, so the teaching of the pseudo-voice progressed and paralleled the advances made in the operation of laryngectomy. By 1909 Gutzman,<sup>17</sup> could report on twenty-five cases of laryngectomy with a good pseudo-voice. Since that time a relatively large number of case reports,<sup>18, 17, 18, 19, 20, 21, 22, 23, 24</sup> on pseudo-voice in laryngectomized persons has appeared. The most prolific recent investigators and exponents of this method of speech production are Seeman,<sup>16</sup> and Hugo Stern.<sup>23</sup> The latter's monumental efforts in this type of endeavor have received recognition throughout the world. Besides a number of articles dealing with speech therapy, his chapter in Denker and Kahler's "Handbuch,"<sup>24</sup> is a classic that should be read by all who are interested in this work. In the United States but little has been written on this interesting and vital subject. Recently, however, Morrison,<sup>25</sup> in an excellent treatise has given a fine review of the work of Seeman and Stern and others.

Our experience with the pseudo-voice has taught us that some patients are unable to acquire intelligible speech. Most of them can quickly acquire a pseudo-whisper rather than a pseudo-voice. This is frequently due to poor instruction or inability on the part of the patient to learn the proper esophageal voice. In production of a pseudo-whisper only a small amount of air is trapped in the oropharynx or buccal cavity. The vicarious glottis is formed between the tongue and palate, or pharynx. In this method true vowels cannot be produced and many consonants but poorly. It is apparent that this type of speech is unintelligible for the most part. Unfortunately, after a patient has acquired this buccal pseudo-whisper it is difficult to teach him a proper esophageal voice.

While we have had a number of patients who could more or less easily acquire an esophageal pseudo-voice, a number could not. In a review of those cases we found that the esophageal introitus was always patent and open. It can be readily understood that, since an esophageal voice depends upon the swallowing and retention of air in the esophagus and the eructation of the air past a constriction at the mouth of the esophagus, formed by the remnants of the cricopharyngeus, a patent esophageal orifice will effectively prevent the production of the pseudo-voice, as the necessary narrowing provided by the sphincteric action of the muscle is absent. Undoubtedly the relaxed condition of the esophageal opening was due to too extensive resection of the cricopharyngeal muscle or poor coaptation, or the severance of the motor nerves at the time of laryngectomy. Since

that time we have used a great deal of care in retaining as much of the cricopharyngeus muscle as possible and respecting the nerve supply to the esophagus in order that this essential part of the vicarious glottis or pseudo-glottis may function as such. Yet, extensive malignant involvement of the larynx may cause us to sacrifice the muscle to a great extent. It follows, therefore, that in a certain proportion of laryngectomized patients, a good pseudo-voice cannot be expected if the patient is psychologically unable to learn, if he has already acquired a buccal pseudo-whisper, or if a functioning cricopharyngeus is not present after the operation.

In 1927 at the meeting of the American College of Surgeons in Chicago, Dr. Joseph C. Beck demonstrated a remarkable case of pseudo-voice. The patient had taken a violent dislike to the MacKenty artificial larynx, although he was an expert in its use. He carefully considered his case and then passed an ice pick, which he had heated red hot, through the opening of his trachea in the neck upwards and backwards until it reached the hypopharynx. He repeated this heroic procedure on two more occasions and subsequently a permanent fistula between the tracheal orifice in the neck and the hypopharynx remained. By placing his thumb on the tracheal orifice and making an expiratory effort with his chest, he was enabled to force a blast of air through the small fistula into the hypopharynx. The passage of the air along the narrow fistula caused the formation of tone, which was conveyed to the hypopharynx and modulated by the usual mechanism into a very loud form of intelligible speech. This man could be heard over a large room with ease. By extending his head he could raise the pitch somewhat as this decreased the caliber of the fistula and placed it under more tension. Further extension of the head closed the fistula altogether and so prevented the ingress of fluids during swallowing. This rather unique occurrence struck the writer that here was the means of forming a tone producing mechanism for those individuals enumerated in the preceding portions of this article, who for one reason or another could not use an artificial larynx or were unable to acquire a competent esophageal pseudo-voice. Several years went by before a suitable case presented itself. In 1931 the following technic was carried out on a laryngectomized patient who was psychologically unable to learn the use of an artificial larynx after months of protracted trial. Due to a relaxed esophageal introitus he could not acquire a good esophageal pseudo-voice. All he was able to learn was a very slight buccal pseudo-whisper that could not be heard over three or four feet and was unintelligible for the most part.

Anesthesia was secured by morphine-scopolamine analgesia supplemented by local infiltration of novocain. An esophagoscope was introduced to act as a guide and prevent the needle from entering the posterior esophageal wall. The needle for the injection of the anesthesia was inserted a few m.m. below the periphery at the upper portion of the tracheal stoma in the neck. Infiltration was continued as the needle was carried upward and backward in the mid-line to just behind the remnants of the epiglottis which had not been removed at the time of the laryngectomy. The needle point was made to emerge posteriorly to the epiglottis in order that that structure by covering the hypopharyngeal opening of the fistula might aid in preventing the ingress of fluids and food while swallowing. The proper course of the needle was effected by using the esophagoscope as a guide. After the anesthetic had taken effect a thin needle electrode attached to a diathermy machine was passed along the same tract, using the guiding esophagoscope until the electrode was seen in the hypopharynx behind the remnants of the epiglottis. The current was then turned on and the needle slowly withdrawn so as to insure an electro-coagulated tract. After a few days the tract began to break down and within two weeks a definite fistula formed.

Two other cases have since been operated on in a similar manner. In one case too much coagulation was used and so a large fistula resulted, through which fluids would trickle while swallowing. In the second case the hypopharyngeal barrier that formed after laryngectomy was so thick that the coagulated tract would heal over. It was finally necessary to insert a thin indwelling urethral catheter to help form a fistula. This last case is still under observation.

Speech can be readily acquired after the fistula has formed. The patient is taught to block the tracheal stoma with his finger or thumb, and then force the air up along the fistulous tract. He easily learns to modulate the resulting tone into speech. By varying the tension on the fistula, by more or less flexion of the head he can also vary the pitch to some extent and so produce a more pleasing and less monotonous voice.

One must acknowledge the fact that an experience limited to but three cases leaves a great deal to be desired. However, laryngectomized patients in whom this operation is indicated are few and far between. The fact that a serviceable voice may be obtained by this procedure, has led to its description in the hope that it may be tried in the hands of others in order that whatever merit it may possess will be more definitely established.

## CONCLUSIONS

1. The social and economic rehabilitation of the laryngectomized patient is dependent upon the physician's ability to endow him with some effectual means of intelligible speech.
2. Many such patients refuse to use an artificial laryngeal prothesis.
3. Many such patients are unable to acquire a serviceable pseudo-voice.
4. A simple procedure is suggested and described that may be of value in voice production in these individuals.

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# A TWELVE-YEAR SUMMARY OF THE TREATMENT OF LATERAL SINUS THROMBOSIS\*

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This report is an attempt to study cases of lateral sinus thrombosis or phlebitis taken from the records of the Massachusetts Eye and Ear Infirmary from 1921 to 1932, inclusive. A definite diagnosis before operation was not made until the sinus was opened. The treatment was the same in cases presenting suspicious findings, and for that reason the diagnosis was always recorded as "Sinus Thrombosis." These cases were operated upon by the various members of the staff. The treatment of both the sinus and jugular vein was, with rare exception, uniformly the same. The procedure was to ligate the jugular vein, expose the lateral sinus freely, incise and remove the sinus wall and the thrombus, when present, until free bleeding occurred, if this was possible. The bleeding was controlled by iodoform packs. These packs were also applied to such cases that did not have a thrombus. Frequently the sinus wall could not be removed when bleeding was profuse.

These cases were divided into two series, the first 1921-1926, inclusive, and the second from 1927-1932, inclusive. A comparison was made of the two series to determine if any progress had been made in the diagnosis and management of such cases. As there is no established standard of reporting data on sinus thrombosis I am presenting such data which seemed of importance to me. No regular attempt is made to compare these findings with the observations of similar cases reported by others. If this were attempted it would lead to a never-ending controversy because of the difficulty in comparing the type of cases of one observer with those of another.

## ANALYSIS OF MASTOIDECTOMIES, ETC.

There were 4,961 mastoid resections which included both simple and radical mastoidectomies and such cases that returned with recurrent infection in mastoids on which operations were previously done. The number of resections is practically equal in both series. The diagnosis of sinus thrombosis for the combined series was 161

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\* Candidate's thesis.

or 3.2 percent. The number of diagnoses of thrombosis in Series II exceeds by almost a third that in Series I. This may have been due to too much conservatism in the first series or perhaps to the fact that the diagnosis on suspicious findings was made too quickly in Series II. Regardless of what may have been the reason for such a diagnosis in Series II, these cases definitely had symptoms which were urgent enough to warrant investigation of the mastoid and sinus.

In both series the males exceed the females. The right side was involved more frequently than the left. This may have been due to the fact that more right mastoids were involved.

The acute cases exceed the chronic cases in considerable numbers in both series. Series II shows a definite rise in the number of chronic cases with the diagnosis of sinus thrombosis.

I might suggest that in the examination of our end-results our percentage of cures compares with the majority of findings reported from the large American and European institutions. It improves somewhat if we exclude the hopeless cases. The hopelessly ill cases on admission in both series were almost equal in number. Those that died within one to three days after admission were excluded in the estimation of our percentage of deaths. Such cases that died on the fourth or fifth day after admission were included in this summary though they appeared hopelessly ill when admitted.

Our cases were certainly not as serious as those in some of the European clinics whose patients often come from far outlying districts where proper medical treatment is not obtainable. This condition exists infrequently in this country as shown by the fewer number of intracranial complications with thrombosis as compared, for example, with Mygind's report of 3,000 mastoid resections in which there were 199 cases of sinus thrombosis, with fifty-three brain abscesses.

The fact that there was a greater number of cases of sinus thrombosis in Series II and that the patients were in the hospital a shorter period of time might indicate a milder type of disease in this series as compared to Series I. Now that the records can be viewed in retrospect I feel that a number of these cases were operated on too early and these patients might have done just as well if the sinus had not been opened or the vein ligated. These milder cases help shorten the average number of days in the hospital for Series II.

The age incidence was interesting. The preponderance of cases of thrombosis occurred between the ages of six and thirty years, the majority of cases appearing between the ages of eleven and twenty years. There were relatively few cases of lateral sinus

thrombosis in very young children compared with the number of mastoidectomies done. The same is true of the very old, but there were relatively fewer mastoidectomies in this class. The infrequency of lateral sinus thrombosis in the very young may be explained on the basis of the position of the lateral sinus which plays a less important rôle in relation to the mastoid cells. Therefore, the more probable pathway of infection is from the hypotympanic space into the jugular bulb. On several occasions in our hospital sinus thrombosis was found in young children without clinical evidence of mastoiditis but only with middle ear pathology. It was felt that the infection went from the tympanic space into the jugular bulb. As the mastoid begins to develop and take form and the sinus becomes well defined in relation to the mastoid cells we see a great rise in the number of cases of thrombosis in children.

SERIES I—1921-1926

<i>Mastoid resections</i>	<i>Sinus phlebitis or thrombosis</i>	<i>Male</i>	<i>Female</i>	<i>Right</i>	<i>Left</i>	<i>Acute otitis</i>	<i>Chronic otitis</i>	<i>All inclusive</i>		<i>Reduced, six hopeless</i>		<i>No. of days' stay in hospital</i>	
								<i>Living</i>	<i>Dead</i>	<i>Living</i>	<i>Dead</i>	<i>Living</i>	<i>Dead</i>
2,496	64	38	26	38	26	48	16	44	20	44	14	50	35
Percent	2.5	59.4	40.6	59.4	40.6	75	25	68.7	31.3	75.7	24.3		

SERIES II—1927-1932

2,465	97	67	30	56	41	58	39	72	25	72	18	35	29
Percent	3.9	69.1	30.9	57.8	42.2	59.8	40.2	74.2	25.8	80	20		

AGE INCIDENCE

SERIES I

SERIES II

<i>Years</i>	<i>Number of cases</i>	<i>Percent</i>	<i>Years</i>	<i>Number of cases</i>	<i>Percent</i>
1- 5.....	4	6.3	1- 5.....	9	9.3
6-10.....	11	17.2	6-10.....	25	25.8
11-20.....	21	32.8	11-20.....	37	38.1
21-30.....	11	17.2	21-30.....	15	15.5
31-40.....	10	15.6	31-40.....	6	6.2
41-50.....	5	7.6	41-50.....	3	3.1
51-60.....	0	0	51-60.....	1	1
61-70.....	2	3.1	61-70.....	1	1

## APPEARANCE OF THE SINUS WALL AND LATERAL SINUS CONTENTS

There were fifty-seven cases in Series I, and eighty-seven cases in Series II in which the records contained a full description of the appearance of the sinus wall and the sinus when incised. It is obvious from this analysis that the appearance and palpation of the sinus wall gives no indication of what may be within it. Some believe the thrill felt when the wall is palpated is an indication that blood is flowing through it. However, this is a difficult sign to elicit when the wall is thickened or when granulation tissue is present. One may see a normal sinus wall in the mastoid cavity and palpation will give the thrill of the eddying blood within it, yet there may be a thrombus low down in the bulbar region. This observation was made on several occasions. For example, when the sinus was incised and free bleeding occurred which was controlled by packing and the incision was continued into the bulbar region, a thrombus was found. Unquestionably, in the presence of perisinus abscess one has more reason to suspect thrombosis or phlebitis. In both series it was surprising to note the number of proved cases of thrombosis with a normal appearing sinus wall. I am inclined to believe

## ANALYSIS OF THE APPEARANCE OF SINUS WALL AND CONTENT OF LATERAL SINUS WHEN INCISED

## SERIES I

<i>Appearance of wall</i>	<i>Number of cases</i>	<i>Free bleeding</i>	<i>Thrombus</i>	<i>Mural thrombus</i>
Perisinus abscess .....	26	7	16	3
Thickened wall .....	11	4	7	0
Granulations only .....	9	5	3	1
Normal .....	11	5	4	2
Total .....	57	21	30	6
Percent .....		37.7	52.5	10.5

Seven incomplete cases not included.

## SERIES II

Perisinus abscess .....	44	17	26	1
Thickened wall .....	14	9	5	0
Granulations only .....	9	5	1	3
Normal .....	20	6	12	2
Total .....	87	37	44	6
Percent .....		42.6	50.5	6.9

Ten incomplete cases not included.



that when this happens it is the result of a retrograde thrombophlebitis in one of the minute vessels, without definite evidence of necrosis anywhere in the surrounding bone.

Analyzing the proved cases of thrombosis it is noted that there were 36 in Series I of which 22 or 61.1 percent were discharged well and 14 or 38.9 percent died. In Series II there were 50 cases with a proved thrombus of which 35 or 70 percent were discharged well and 15 or 30 percent died. In regard to those with free bleeding, in Series I there were 21 such cases; of this number 15 or 71.4 percent were discharged well and 6 or 28.6 percent died. In Series II there were 37 cases with free bleeding. Twenty-seven or 73 percent were discharged well and 10 or 27 percent died.

At this time I might report that there were 141 cases of perisinus abscess with mastoiditis in which complete exposure of the perisinus abscess resulted in the abatement of all symptoms and a convalescence without complications. The symptoms were never sufficient to suggest the possibility of sinus thrombosis.

#### COMPLICATIONS

The variety of complications associated with sinus thrombosis could be divided into intracranial and other types. By complications I mean something directly associated with the ear infection. All of our meningitis cases where organisms were found in the spinal fluid died. Six meningeal cases presented the following signs: cloudy fluid under pressure, stiffness of the neck, positive Kernigs, and other signs of meningeal irritation, but the spinal fluid showed no organisms nor was the sugar reduced or the Pandy test positive. However, from the temperature and other clinical signs there was sufficient evidence to suspect a meningitis. These cases all got well. It is interesting to note that there were only six such cases in these series. For a long time I have been under the impression that these meningeal signs occurred more often in cases of proved thrombosis, particularly, if the thrombus was infected and had extended to the inner or cerebellar wall of the lateral sinus. There is no question that it does occur but not as often as one would suspect. In the absence of definite intracranial complications but in the presence of meningeal signs without organisms in the spinal fluid one must continue to examine the sinus as a cause of these symptoms.

Three of the brain abscess cases in Series II got well. Considering the small number of cases of brain abscess complicating sinus thrombosis it would seem that these cases have been benign. This



## OTHER COMPLICATIONS

Facial paralysis 1	Facial paralysis 2
Kidney { Perinephritic abscess 1	Gradenigo syndrome 2
{ Acute hemor. nephritis 1	{ Pneumonia 5
Lung { Pneumonia 1	Chest { Pulmonary infarct 1
{ Pulmonary infarct 1	{ Multiple lung abscess 1
Osteomyelitis, femur, 1	{ Empyema 3
	Kidney, acute hemor. nephritis 3
	Osteomyelitis { Humerus 1
	{ Femur 1

## METASTASIS

In Series I we found that metastasis occurred in 21.8 percent of the cases and in Series II in 31 percent. It was by far more commonly present in acute otitis than in chronic otitis. Also it occurred more frequently in those cases with a proved thrombus. Metastasis appeared with much greater frequency, in fact about four times as often, after ligation as before ligation. The most common complication of sinus thrombosis unquestionably is involvement of the joints. All seemed vulnerable, the hip, ankle, knee, shoulder and others in order of relative frequency. Many of these metastatic foci required surgical drainage. In Series I six patients were operated upon for one or more foci, and in Series II ten such patients were drained surgically. Metastasis, as a complication of sinus thrombosis, though not of grave consequence, adds to its seriousness.

It was noted that several cases in both series had metastasis before ligation as well as after ligation. Seven patients had single metastatic foci and seven others multiple foci, more than two or three areas being involved, in Series I. In Series II nineteen patients had single metastatic foci and eleven multiple foci, one having as many

## METASTASIS

	No. of cases	Acute otitis	Chronic otitis	Metastasis		Well	Dead
				Before lig.	After lig.	Acute and chronic	Acute and chronic
Series I .....	14	12	2	3	11	10*	4†
Percent .....	21.8			21.4	78.6	71.4	28.6
Series II .....	30	22	8	7	23	22‡	8§
Percent .....	31			23.4	76.6	73.2	26.7

\* Acute 9, chronic 1. † Acute 3, chronic 1. ‡ Acute 19, chronic 3. § Acute 3, chronic 5.

as four. In Series I of the three cases that had metastasis before ligation two developed foci after ligation, so that thirteen out of the fourteen cases had metastasis after ligation. In Series II of the seven that had metastasis before ligation, three had foci after tying the vein, so that of the thirty cases twenty-six actually had metastasis after ligation.

APPEARANCE AND FINDING AT INCISION OF LATERAL SINUS  
IN METASTASIS CASES

SERIES I

<i>Appearance of sinus wall</i>	<i>Number of cases</i>	<i>Thrombus</i>	<i>Free bleeding</i>	<i>Metastasis</i>	
				<i>Before ligation</i>	<i>After ligation</i>
Perisinus abscess .....	9	5	4	2	7
Granulations or thick wall	3	3	0	1	2
Normal .....	2	1	1	0	2
Total .....	14	9	5	3	11
				2 F. B., 1 Th.	3 F. B., 8 Th.

Distribution: Hip 7, ankle 5, shoulder 5, knee 3, elbow 1, sternoclav 1, femur 1, kidney 2, lung 1.

SERIES II

Perisinus abscess .....	15	10	5	3	12
Granulations or thick wall	8	6	2	2	6
Normal .....	7	5	2	2	5
Total .....	30	21	9	7	23
				3 F. B. 4 Th.,	7 F. B. 16 Th.,

Distribution: Hip 7, angle 5, knee 3, elbow 3, sternoclav 2, shoulder 1, foot 2, thigh 2, scapula 1, arm 1, kidney 3, chest 6.

ANALYSIS OF FATALITIES

One would expect that sepsis in chronic otitis media would be more serious than in acute otitis, because, it seems, more severe complications occur in the chronic cases. In Series II there were sixteen fatal cases with chronic otitis as compared with nine with acute otitis. The figures are reversed in Series I. Here the fatalities with acute otitis greatly exceed those with chronic otitis. When septicemia is complicated by metastatic foci the prognosis is more grave, as illustrated by Series I, in which four of the twenty fatal cases had metastasis, and by Series II, in which there were eight cases of fatal metastasis.



## THE NUMBER OF DAYS ELAPSED AFTER MASTOIDECTOMY BEFORE LIGATION OF JUGULAR OR OPENING OF SINUS

Boies described three types of sinus thrombosis in a review of the records at the Massachusetts Eye and Ear Infirmary: (1) manifest, with symptoms of thrombosis or phlebitis at the time of operation, (2) latent, not giving any indication that thrombus was present until some days after the mastoidectomy was done, (3) postoperative, those which were probably due to the simple mastoidectomy, wherein an exposed sinus in contact with infected secretions during convalescence became infected, thereby favoring the formation of a thrombus.

In this analysis I feel that sinus thrombosis in postoperative cases is an infrequent complication, and that latent sinus thrombosis occurs more frequently by comparison. The diagnosis of sinus thrombosis at the time of the initial operation exceeds by far the other types.

There is always the danger of error in diagnosis when one tries to complete the entire operative work on the same day because of the presence of a high temperature and a chill. These symptoms may be nothing more than an indication or a warning of the infection. Evidently these symptoms occurred frequently in Series II and were promptly treated, as shown by the great number of cases of free bleeding as compared with the number of proved thromboses in this series.

### AYER-TOBEY TEST

The accuracy of this test in our series was somewhere between 75 to 80 percent. In Series I there were only 11 reports as this test was just coming into use at the Infirmary. In Series II there were 53 tests. In Series I the manometer readings were completely verified in 72.7 percent; partly verified, in that a mural thrombus was found, in 9.1 percent; and unverified in 18.2 percent. In Series II the manometer readings were verified in 64 percent; partly verified in 9 percent; and unverified in 27 percent. It continues to be one of our favored diagnostic aids in lateral sinus thrombosis.

### BLOOD CULTURES

I feel that when the blood culture is negative an operation should not be postponed, particularly if the symptoms are of such proportion that investigation of the sinus is indicated. If the patient is not losing ground consistently negative cultures warrant conservative action. A positive culture before mastoidectomy is of value in that it points to invasion of the blood stream, occurring most likely

through the lateral sinus and calls attention immediately to that region. I do not, on the other hand, recommend immediate action on the lateral sinus because the blood culture is positive.

In this analysis I am utilizing only the blood culture reports in Series II. Sixty-five patients had complete blood reports, from two to six cultures being made on the same patient. The blood culture was negative before ligation fifty times, in thirteen of which it became positive after ligation, tending to indicate that tying of the vein does not prevent bacterial invasion of the blood. Surprising as it may seem, of the fifty cases that were negative before ligation thirty actually had a thrombus present when the sinus was incised.

#### ACCIDENTAL INJURY TO THE SINUS

There were six cases of accidental injury to the lateral sinus which gave rise to symptoms requiring ligation of the jugular vein and incision of the sinus. At the time of the injury the sinus wall was normal in four and superficially infected in two of the cases. When injured all bled freely. Metastasis did not occur in any of these cases. One patient died and it is reasonable to assume that the death was due to the accident as there was no evidence that he had septicemia before the injury. On the other hand one cannot be sure that he might not have developed sepsis postoperatively. During this twelve-year period the sinus wall was injured on several occasions but nothing happened to indicate that the trauma was harmful; the patients were free from symptoms and convalesced in the usual manner. One patient developed a cerebellar abscess which was drained through the posterior sinus wall. This patient recovered.

#### ANALYSIS OF FATAL CASES

SERIES I  
1921-1926

		Thrombus	Free bleeding
Meningitis .....	9 { 3 chronic 6 acute	2 5	1 1
Septicemia .....	8 { 1 chronic 7 acute	0 5	1 2
Brain abscess .....	2 { 1 chronic 1 acute	1 1	0 0
Pulmonary embolus .....	1 1 acute	0	1
Total 20 cases	{ 5 chronic—25% 15 acute—75%	{ 3 thrombus 2 free bleeding 11 thrombus 4 free bleeding	

ANALYSIS OF FATAL CASES—*Concluded*

## SERIES II

1927-1932

Meningitis .....	6	{ 5 chronic 1 acute	3 0	2 1
Septicemia .....	11	{ 7 chronic 4 acute	4 2	3 2
Brain abscess .....	3	{ 1 chronic 2 acute	1 2	0 0
Chest { pneum. and emp. ....	2	{ 2 chronic	2	0
{ pneumonia .....	2	{ 2 acute	1	1
{ mult. lung abs. ....	1	{ 1 chronic	0	1
Total 25 cases	<div style="display: flex; align-items: center;"> <div style="margin-right: 10px;"> { 16 chronic—64% 9 acute—36% </div> <div> { 10 thrombus 6 free bleeding 5 thrombus 4 free bleeding </div> </div>			

NUMBER OF DAYS ELAPSED AFTER MASTOIDECTOMY BEFORE LIGATION  
AND OPENING OF SINUS

## SERIES I

<i>Same day or 1 to 2 days</i>		<i>3 to 7 days</i>	<i>8 to 14 days</i>	<i>15 days or more</i>			
28	$\left\{ \begin{array}{l} 20 \text{ thrombus} \\ 6 \text{ free bleed.} \\ 2 \text{ inc. data} \end{array} \right.$	16	$\left\{ \begin{array}{l} 8 \text{ thrombus} \\ 7 \text{ free bleed.} \\ 1 \text{ inc. data} \end{array} \right.$	14	$\left\{ \begin{array}{l} 6 \text{ thrombus} \\ 6 \text{ free bleed.} \\ 2 \text{ inc. data} \end{array} \right.$	6	$\left\{ \begin{array}{l} 2 \text{ thrombus} \\ 2 \text{ free bleed.} \\ 2 \text{ inc. data} \end{array} \right.$

## SERIES II

65 { 38 thrombus 22 free bleed. 5 inc. data	21 { 9 thrombus 9 free bleed. 3 inc. data	5 { 1 thrombus 4 free bleed.	6 { 2 thrombus 2 free bleed. 2 inc. data
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## TREATMENT

The procedures recommended for the treatment of sinus thrombosis vary greatly and yet the adherents of each method claim an approximately equal percentage of cures. If this is true is it not reasonable that we should make use of the procedure which accomplishes as much with a minimum surgical interference? From these records the majority of undesirable complications occurred after ligation. If ligation seemed to do no harm it certainly could not be proved that it aided the patient. Why then add ligation to the surgical treatment of lateral sinus infection? There is always the risk of a secondary infection in the neck wound and injury to the vein and nerves in that region. These structures have been injured even by the best technicians.

I feel that the thrombus should not be completely exenterated. It is imperative that all diseased bone overlying the sinus should be removed, carrying the operation into the deepest confines of the mastoid if necessary. The sinus wall should then be incised and as much of it removed as possible, and the mid-portion of the clot cleaned out sufficiently to expose the inner wall of the sinus, the area lightly packed with iodoform and the mastoid cavity left open for drainage. If the clot is too small to permit this procedure it should be exposed without establishing free bleeding and packed as described. Should free bleeding occur where a thrombus was expected there is little that one can do except to place the iodoform packs over the incision and control hemorrhage. Further treatment then depends on the symptoms.

Obviously, one cannot lay down a hard and fast rule for the treatment of all cases of sinus thrombosis. One should evaluate the signs and symptoms and treat the case as the occasion demands. One has no right to assume that the worse may happen if one treats conservatively. The indications for an extremely radical procedure in this series were rare, and when performed it failed to influence the outcome. In the event of a fatality, if reasonable care was exercised one should not be troubled because he did not carry out every known surgical procedure. The end-result is due to the ability or inability of the individual's resistance to cope with the virulence of the infection.

It must be added that the comment made on this review, particularly in regard to the treatment, does not represent the consensus of opinion of the members of the staff of the Massachusetts Eye and Ear Infirmary. It is entirely a personal opinion. Further, I do not wish to give the impression that the treatment is in anyway an original one, as it has been proposed for many years by numerous surgeons in this country and abroad. It may vary a little in detail but the fundamental principles are unchanged.

I also wish to state that, undoubtedly, there will be a rare occasion which will demand treatment of the jugular vein. Should symptoms arise which demanded this treatment I would not hesitate to apply it.

Summarizing this review I noted the following:

1. Symptomatically it is impossible to distinguish between sinus thrombosis and phlebitis.

2. In a total of 4,961 mastoid resections the diagnosis of sinus thrombosis was made in 161 patients indicating that it occurred once in approximately 31 cases. It is the most frequent complication of



mastoiditis, the greater number occurring in acute otitis. It occurred more often in the ages between 11 to 20 years, tapering off sharply at both age extremes.

3. Metastasis was an uncommon complication before ligation of the jugular vein. Involvement of the joints was the most common complication. The greatest number occurred after ligation and treatment of the sinus. It adds to the seriousness of the disease as it may provoke the focus which continues to feed organisms into the blood stream.

4. Blood cultures are of value as an aid in establishing a diagnosis or at least in focusing attention on the lateral sinus. Negative blood cultures in the presence of continuing symptoms should not deter from examination of the sinus.

5. The Ayer-Tobey test is one of the valued diagnostic aids. The reliability of the test is dependent on experience and carefulness in its performance.

6. The appearance of the sinus, from these records, bears out definitely what is commonly known, that it gives no indication of what may be within it. One has more reason, however, to suspect injury to the endothelial lining in cases with perisinus abscess.

7. Sinus thrombosis is not necessarily a fatal disease if untreated surgically. Surgery is always indicated in my opinion as the best means of aiding the patient to overcome his infection, affording drainage and thereby tending to limit the disease. There is no such thing as extirpation of the diseased focus. We aid the patient in the disease but the battle for supremacy is between the infecting organism and the individual's resistance.

8. Postoperative sinus thrombosis occurs infrequently as compared with the manifest and latent types.

9. Secondary infection of the neck wound occurred quite commonly.

10. The percentage of fatalities in general may be considered to be between 20 and 30. In this report are excluded the hopelessly ill on admission. In the combined series there were 148 of which 32 or 21.7 percent died and 116 or 78.3 percent got well. Excluding intracranial disease sinus thrombosis must be considered to have a relatively high mortality.

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# RELATIONSHIP OF CLIMATE TO DISEASES OF THE UPPER RESPIRATORY TRACT\*

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## PART I

### PHYSIOLOGY OF THE UPPER RESPIRATORY TRACT

Huntington, in his work entitled "World Power and Evolution," emphasizes man's dependence on climate, and avers that it has been the most important factor in his evolution. The human body is dependent upon atmospheric environment and, by the respiratory apparatus, it adapts itself to variations of temperature and humidity. Nature in its wisdom has fashioned the body so that it is particularly susceptible to the influence of the agent or medium to which it is adapted. Therefore, it is only reasonable to assume that the respiratory tract is sensitive to abnormal atmospheric conditions.

Many centuries ago Hippocrates wrote after this fashion:

Whosoever wishes to investigate medicine properly, should proceed thus. In the first place, consider the seasons of the year, and what effect each of them produces, for they are not all alike, but differ from themselves in regard to their changes; then the winds, the heat, the cold, such as are common to all countries, and such as are peculiar to each locality.

We consider first the physiology of the nose, whose function is, as a respiratory organ, to heat, moisten and filter the air and to act as a protector against the inspiration of bacteria. In the Polar regions it warms the air to such an extent that it removes danger of freezing lung tissue. It gives off moisture at the rate of six ounces in twenty-four hours, which acts as a protective film over the mucous membrane and helps maintain water balance in the body. The nose also acts as a filter by removing dust from the inspired air and secreting a thin, albuminous liquid which serves as a protection against the entrance of bacteria to the nasal mucosa. This heat and moisture mechanism of the body is controlled by the vasomotor system of the upper respiratory tract and the skin.

Heat dissipated through the skin (approximately 80 percent) is given up by evaporation, convection and radiation. Heat given up by the nasal mucous membrane (approximately 20 percent) is largely

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\* Candidate's thesis.

by radiation. In this way the skin regulates the internal temperature and keeps the surface of the body warm, while the nasal mucous membrane serves the function of warming the air for the respiratory organs.

For the patient who lives in a cold, damp atmosphere, such a process accounts for decreased moisture eliminated by both nose and skin, with a resulting nasal stasis; while the patient who lives in warm, dry air, loses moisture by the skin to such an extent that an insufficient supply of moisture exists in the nose to make it function properly, and nasal dryness is the result.

Warm, moist air (about 50 percent humidity) would result in the nasal mucosa being properly moistened, and at the same time enough moisture would be eliminated by the skin to keep the water balance normal and avoid nasal stasis.

Thus, we can see how low winter temperature with accompanying high humidity predisposes to infections of the upper respiratory tract more easily than high summer temperature with low humidity. When the body is exposed to air, either damp or dry, of a temperature and humidity that produces a loss of heat from the body faster than the heat mechanism is able to produce heat within the body, a chill results. This heat production mechanism, the writer believes, is affected by the presence of infections, *i.e.*, an infected person will not produce body heat to such a great extent, due to subnormal metabolism, as a healthy person and is, therefore, susceptible to slighter variations of temperature. Also, it is noticeable that a higher temperature in which to live comfortably is required of the older than the younger generation.

Dry air, as studied by the Department of Public Health in Chicago,<sup>1</sup> causes irritation in the mucous membrane, sleeplessness, irritability, fast pulse and dryness of skin. Chronic bronchitis is aggravated by irritation. Dry air also causes burning of the eyes, due to rapid evaporation of secretions of lining membranes.

Too much humidity interferes with proper evaporation and dissipation of body heat, thus accounting for great prostration, as when Chicago was reported to have a temperature of only 88 degrees about July 17, 1934, with humidity of 88 percent, which resulted in many heat prostrations. At the same time San Bernardino, California and Phoenix, Arizona, frequently showed temperatures from ten to fifteen degrees higher with low humidity of around 30 percent, and with few, if any, heat prostrations. Thus, we are convinced that excessive moisture makes hot air feel hotter and cold air feel colder.

Consequently, air which is too moist for its accompanying temperature is unhealthful. Many writers attribute the same effect to very dry air, but this writer believes this statement should be qualified to apply to the condition of air within buildings, while the air on the desert rarely ever contains enough moisture at ordinary temperatures to be prostrating, and becomes intolerable only at low humidity and when the temperature reaches over 110 degrees in the shade.

Mention should be made of the allergic patients who are sensitive to the dust that is frequently present in dry desert air. These sensitive patients find the desert very uncomfortable as a result of this irritant.

In a paper by Dean Blake,<sup>2</sup> "Some of the Functions of Humidity," the author states: "The idea that the thermometer alone registers the heat and cold experienced by the human body is entirely too prevalent, judging by the opinions of health seekers who examine data in the weather bureau offices. Other climatic elements such as wind movement, rate of evaporation, and the amount of water vapor in the air also affect our bodily sensations of heat and cold. Severe cold, for instance, becomes unendurable when accompanied by wind, and calm weather with a hot, muggy atmosphere is also productive of great discomfort, and even fatalities.

"Moist air has greater capacity for heat than dry air, providing the temperature is the same. At 32 F., moist air 'feels' colder than dry air, because under these conditions, it takes away heat from the skin faster and leaves the skin colder. When the temperature is higher the heat is kept in the body by the humid air because, owing to poor conduction and radiation, evaporation is hindered and heat loss is prevented.

"Most writers agree that damp air induces nervous depression, slower blood circulation and quiet sleep, while dry air acts as a nervous excitant and causes sleeplessness and restlessness."

In a paper by Daniel Weaver<sup>3</sup> on "Why Colds?" published in the *Journal of Indiana State Medical Society*, he states in part:

The mucous membrane of the whole upper respiratory tract and lungs secretes about one and one-half pounds of water each twenty-four hours in a normal atmosphere. This is for protection of the delicate epithelium, just as the lachrymal gland secretes a fluid to protect the epithelium of the cornea, without which the cornea would be destroyed in forty-eight hours. If, through external or internal influences, or both, the moisture is not maintained over the mucous membrane, destruction of the epithelium follows.

All things that come in contact with the mucous membrane of the nose, whether intentionally or inspired through the air, and which are irritating to the membrane and cause corrosion or exfoliation of the surface epithelium, are potent factors in the production of colds.



Dust particles in the air, especially those of soluble lime, are exceptionally irritating and cause corrosion. A sudden change in the temperature of the air we breathe, fumes of noxious gases, and excessively dry atmosphere are destructive forces.

The lime dust of a Sunday afternoon automobile traffic is destructive to the epithelium cells, and the bacterial flora present at all times gain a foothold in the denuded surface—result, a cold. The same is true of irritating dust in sweeping with a broom, or the dust behind the threshing machines and that arising in various dusty vocations. But the most serious source of injury to epithelial covering of the mucous membrane is drying, due to low humidity of the atmosphere in artificially heated homes, public halls, schools and places of business. The effect is well illustrated in the epidemics of colds among school children when fall heating begins. The upper respiratory tract cannot protect itself, except for a short time, against bacteria or irritating foreign matter in an atmosphere of low humidity. The membrane becomes too dry to function properly as a filter, thermostat, or humidifier, and the bacteriocidal function is practically destroyed. The surface epithelium in low humidity is exposed to the destructive influence of dryness and foreign matter in the atmosphere which it cannot resist indefinitely. The destruction is followed by bacterial infection—colds.

In order that the nose may function normally, the atmosphere indoors should be of a humidity of 50 percent saturation, and a uniform temperature of 68 to 70° F. Any marked variation from this humidity and temperature is an agent for respiratory infection.

In a paper read by A. Lowndes Yates,<sup>4</sup> on the "Activity of Ciliary Epithelium," he makes the following two conclusions:

*First*, that the severity of a sinusitis, or sinus infection, depends upon the power of the mucous membrane to prevent its destruction by the inspired micro-organisms, and if the property that mucus possesses of preventing the passage of water and hence of water-soluble toxins is destroyed, that a progressively increasing poisoning by these toxins of the ciliated epithelium occurs.

As the ciliary paralysis increases in amount, the secretion of mucus is also paralyzed until finally the micro-organisms come to dwell in a symbiotic state in the sinuses, and the toxins from them are readily absorbed with the production of remote symptoms.

*Second*, that the intimate relation of the tracts from the sinuses to the Eustachian tube suggests that Eustachian catarrh is a secondary manifestation of sinusitis, and that the intimate relation of the tracts from the sinuses to the tonsil suggests that the initial enlargement of the tonsil is frequently due to sinusitis, although the tonsillar enlargement may be due, of course, to other causes.

McDonald, Leisure and Lenneman<sup>5</sup> have pointed out that ciliated epithelium, lining of the sinus, exerts a certain bio-physical defense against invasion of pathological bacteria and that this ciliary action might be augmented or retarded and local resistance raised or lowered. Among the factors augmenting ciliary action was temperature; warm-

ing increased and raised resistance, while cooling definitely retarded its action and lowered resistance.

The effect on persons exposed to heat and very low humidity is a form of prostration with considerable dehydration. These patients evidently lose more chlorides than they should, since the taking of larger quantities of chlorides (especially sodium chloride) tends to prevent this type of prostration. Saline given intravenously proves to be the best means of restoring them. These facts suggest a corresponding opposite condition where insufficient sweating or loss of fluid by the skin occurs and prompts us to ask if a lessened sodium chloride intake might be indicated.

Ullman<sup>6</sup> in his paper, "Surgery and Dietetic Treatment and its Relation to Nasal Pathology," discusses the relationship between chlorides and calcium.

Mention should be made of the variation in sensitivity of patients, *i.e.*, at one extreme we find in atrophic rhinitis the hypo-sensitive patient who will permit, without discomfort, the placing of applicators, tampons or Eustachian catheters. At the other extreme, there is the hyper-sensitive rhinitic who experiences great discomfort from the smallest amount of mechanical manipulation. Thus, we understand why one person may go in the ocean or through a dusty desert or through pollen laden air without much ill effect, while the hyper-sensitive patient develops a severe rhinitis by doing so.

## PART II

### EFFECT OF CLIMATE ON THE INCIDENCE OF HEADCOLDS

The Pickett-Thomson Research Laboratory of London conducted an extensive review of over 2,000 research papers on the common cold. The authors who, themselves, have done careful research on the common cold for fifteen years, do not hesitate to say that the incidence of the common cold is almost entirely a matter of climate.

On the other hand, W. H. Barrow<sup>7</sup> in the study of the incidence of colds among students at universities located in different parts of the United States, found that at Stanford University in California where the climate is mild, practically the same number of students for each hundred investigated suffered from acute colds as did those at Harvard University in Massachusetts.

Townsend,<sup>8</sup> of the United States Public Health Service, conducted a study of a large group, mostly of students of colleges, and of another group composed of medical officers of the Army, Navy and Public Health Service and members of university faculties. The

universities were scattered as far as Boston, New Orleans, Chicago, Tucson, Salt Lake City and Berkeley, and the reports covered a period of more than eighteen months. The incidence of colds at the ten universities was found to be remarkably uniform. In fact, the uniformity of attack rate was one of the most significant facts brought out by this study.

On the question of humidity in relation to respiratory diseases, R. M. Hutton,<sup>9</sup> Senior Investigator, Division of Industrial Hygiene, Ontario Department of Health, states in part:

Comparison between cotton and woolen industry is of interest. The spinning, doubling and weaving of cotton are carried on in conditions of artificial heat and moisture, whereas the similar processes of wool are not. The mortality from respiratory disease among the cotton workers is well above the average, while that among the woolen workers is well below. From bronchitis the cotton spinners suffer a mortality nearly three times as great as the wool spinners; the doublers about two and one-half times as high and the weavers nearly twice as high. A similar analysis in cotton weaving alone shows that the mortality from diseases of the respiratory system (bronchitis and pneumonia) of weavers in wet sheds is something like three times that of the weavers in the dry sheds. In these cases, therefore, it looks as though artificial heat and humidity had a causative relation to respiratory mortality. . . .

In a paper by Hilda M. Woods,<sup>10</sup> Division of Epidemiology and Vital Statistics, London, the question of relationship between the unfavorable climatic conditions and the death rate from respiratory diseases is discussed and the seasonal curve of mortality is observed . . . . The winter rise in mortality from respiratory diseases corresponds to the fall in temperature, and with the rise in temperature in the spring there is a decline in the respiratory death rate. But the month of maximum mortality is not in the depth of winter but in February—the time of greater variation.

This corresponds with the experience of numerous physicians practicing in the neighborhood of the Great Lakes. Here respiratory infections occur in the late autumn and early winter, decrease through steady cold weather, or mid-winter, and again recur to a peak during the months of March and early April. The work of many physicians during these "peaks" would show two or three times the number of visits and treatments to patients suffering from respiratory diseases than at any other time of the year. One should note the climatic combination of humidity and marked changes in temperature, *i.e.*, between midnight and midday. During March and November the combination of rain, muddy streets and roads, freezing nights and thawing days, seems to be about the most potent factor predisposing to colds, in the writer's experience.

In a study by Russel of two districts of two metropolitan boroughs, Islington and St. Pancras, over a period of twenty-one years, he concluded that fog itself has no appreciable effect on the death rate *unless associated with a low temperature and frost*, and the effect on the mortality of young children is not so great. He illustrates this by selecting two periods of great fog (1883-84 and 1892-93). In the former period there was no obvious increase in mortality after the fogs. In the latter period there was a large increase. However, in the former, there was no associated sharp fall of temperature, as occurred in 1892-93. The conclusion is that it is essential to bear in mind that, in any study of the influence of weather on the prevalence of disease, the assignment of the increased mortality to any particular factor is not so simple as is generally believed. The fact, often overlooked, is that there are a multiplicity of causes, all of which are highly inter-related, and it is the cumulative effect of these, and not the influence of an individual factor, that finds expression in an increased death rate from respiratory disease.

Frederick N. Sperry,<sup>11</sup> in his paper "The Common Cold," mentions the fact that Government statistics would indicate that the mortality in respiratory diseases, especially influenza, is greater where there is great humidity and severe cold. Moderately cool and relatively dry weather has been marked by lessening colds. The geographical studies of Elsworth Huntington of Yale are at some apparent variance with these statistics. His studies, however, have more reference to civilization and general health. The hardening effect of cold has been noted in Arctic explorers and in woodsmen who tramp in the snow and are frequently wet and fatigued, yet are free from colds. The records of the Metropolitan Life Insurance Company show that in winter indoor, sedentary workers have nine times as many colds as taxi drivers and four times as many as soldiers in the United States Army. . . .

It is also necessary to consider the sun as a climatic factor. V. S. Cheney,<sup>12</sup> Medical Director, Armour & Co., Chicago, says in part regarding this subject: "During the winter we lose a part of the actinic effect of the ultra-violet rays, due the sun's southern declination which requires the rays to pass through a thicker stratum of atmosphere which, especially if smoke-laden, acts as a filter to the rays and materially decreases their chemical action upon the blood, chiefly influencing its calcium content or calcium fixation." As proof of this fact, we point out that skin infections, such as impetigo, which flourish in the winter, will clear up in summer.



Gilchrist and Wilson,<sup>13</sup> writing on "Staphylococcic Infections in Diabetes Mellitus," say that in their observations for the past five years they have brought out the fact that in a large number of diabetic patients they were dealing with a staphylococcic infection of the upper respiratory tract. Their conclusion is that focal infection inhibits the normal production of insulin and that the condition is made worse by damp weather.

### PART III

#### CLIMATE AND NATURAL HISTORY

Until the advent of modern bacteriology, climate has always been regarded as a potent factor in the causation of colds. However, since the discoveries of Pasteur the tendency has been to relegate to the background the old thought and our inclination to over-emphasize one particular factor has caused us to forget many other important points, of which climate is only one. We have thought, in the past, that inasmuch as a draft is only fresh air it is impossible to derive any ill effects therefrom, and overlook the fact that this over-dose of fresh air has the ability to abstract more heat from the body than it can afford to lose, thus resulting in a chilling effect, especially if the body is at rest, when heat production is at its lowest point. If we choose to prove these points by experiments we might refer to Lode,<sup>14</sup> who infected his animals with pneumococcus and shaved one-half of the body and, after a warm bath, exposed them to an open window. The result was infiltration of the lungs, sometimes fatally. Jezerski's<sup>14</sup> experiments differed somewhat in that they were milder and more nearly reached real experiences. He did not use such severe cooling methods and did not withdraw nature's method of protection by shaving the animals. He found that by cooling an already moist, uncovered part of the chest in the case of rabbits or goats, there always occurred some changes in the lungs from simple engorgement to more marked disturbance.

Slight changes from moderate to lower temperature appear to have a stimulating effect on metabolism, while too great or too sudden changes produce chilling which results in lowered resistance and the invasion of organisms, resulting in the onset of colds, and other respiratory diseases.

Temperature rises ranging from moderate to severe, coming on suddenly, appear to be enervating, lowering resistance considerably and in some patients permitting the light-up of infections that are otherwise arrested. Thus, southeast wind of the Hawaiian Islands

and the warm, wet seasons along the Zambezi bring on the dreaded tropical fevers.

In the temperate zones most domestic and wild animals provide for the onset of winter with more elaborate hair or fur and large quantities of fat. The bear, for instance, prepares to hibernate by growing a very furry covering and gaining weight. Mention should be made that fur bearing animals are always marketed in winter when the fur is in its finest condition. Even man in cold climates has a tendency to gain weight up to the onset of winter and usually maintains this extra weight until spring, illustrating the stimulating effect of cold on metabolism.

Among migratory birds the wild goose of the North migrates far to the South when winter comes and remains there until Spring. One can understand why it flies South, but why does it return to the North in the spring? Wild life is concerned with food, cover and climate. These necessities can be obtained in many climates. Why does all migratory bird life move North when spring comes? Why does the barren land caribou make such long migrations? Why does the salmon cross the Atlantic? Why does the eel travel from the West Indies to the shores of Northern Europe? If we study these natural history problems I believe we will be obliged to conclude that climate and principally temperature, modify metabolism so greatly that instinctively all wild life is affected by it. The largest bear lives in Alaska. The largest moose lives in the Yukon. The largest tigers do not come from India but from the rugged, mountainous district of Western China. The largest of elephants, the African elephant, seeks the highest plateaus in the mountains separating British East Africa from the Belgian Congo, at an elevation of 7,000 feet. The greatest ape, the gorilla, makes his home in this district.

Similarly, we might trace the wanderings of the human family, and be able to show that they have preferred to seek climates where marked seasonal changes occur, in other words, where they have enjoyed the most physical benefit. History reveals the fact that most of the world's conquering races have lived in temperate rather than torrid zones.

The effect of climate on the Canadian Cavalry forces sent to South Africa during the Boer War about 1900, namely, Lord Strathcona's Horse, is of interest. An officer on his return told the writer that these animals stood the change of climate very poorly, notwithstanding the fact that they had been range horses, or bronchos, and were considered very hardy animals in their Western Canadian home. He stated that six months after they arrived on Orange Free State

and the Transvaal scarcely a single head remained alive. This was attributed to change of climate, food and water.

A similar example is the effect on the Eskimo when removed from the Arctic Circle, as experiments carried out by both United States and Canadian Governments have shown. Of course immunity and the effects of civilization are also factors, as well as climate. The same applies to officers of the British and American Army and Naval Service in the Tropics. In some instances four years is the maximum of their tolerance.

The writer has observed over a period of fourteen years that hemoglobin percentages, in the hospitals in which he works in Southern California, seem to run uniformly lower than in the North. Tonsil operations require more careful attention. In the writer's opinion, hemostatic pressure after the operation is not enough in many instances, but more care must be taken to ligate bleeders than is the custom in the North. This applies more particularly to adults.

Dr. Clarence A. Mills,<sup>15</sup> professor of experimental medicine, University of Cincinnati, states in part:

Moist heat that depresses bodily metabolism makes for an increased susceptibility to infection and lessened ability to fight bacterial invasions, once they have gained a foothold.

I have found definite differences in this respect between the more active people of the North and the Southerners of more sluggish metabolism. Of each 100 cases of acute appendicitis handled in hospitals, the fatality is almost three times as high in the South, with a steady lowering of the fatality rate as one goes northward from the Gulf region.

#### PART IV

#### CLIMATE AND TUBERCULOSIS CASES

No disease of the respiratory system has been more obstinate to treat than pulmonary tuberculosis. Medicine has long since proved worthless and treatment was given up as being useless for a time, until the advent of rest, open air and diet. The writer has for years been interested in the effect of climate on these patients and has observed that at the onset it was customary to send patients to the Pacific Southwest, probably on account of lower humidity, less extremes in out-door temperature and more hours of sunshine. Undoubtedly, like many other new ideas, climate was over-stressed and many fantastic reports were made. The writer has tried to get the truth on this particular subject and has been told by several eminent chest specialists that climate does not amount to more than 10 percent as a therapeutic measure.

There are features about this subject which have proved interesting. First, why do advanced cases of this disease, when arrested in these Pacific Southwest climates, have to remain there? Experiments have been made on average healthy Eastern students coming to Colorado Springs. Complete blood counts have been done on their arrival and again at the end of three months when they departed for the East. These blood counts and hemoglobin estimations showed uniformly marked increase in cell count and percentage of hemoglobin over control cases which had not visited Colorado Springs, but remained in Eastern cities. Again, these patients whose cases have been arrested, and who remained where they were cured, remain arrested a much longer period of time than those who return to their original locality. Why is this? The writer is not concerned with any particular angle of this subject other than the scientific and is aware that most tubercular patients are treated at home regardless of climate, and are arrested with average good results. But, having visited several centers such as Denver, Colorado Springs, El Paso, Tucson and Phoenix and consulted patients coming from all over the United States and Canada, the writer has been impressed with the uniformity of the personal history of these people, *i.e.*, that nearly all made more rapid progress in the Pacific Southwest, and those that had come too late usually expressed their regret.

V. O. Knudsen,<sup>16</sup> of the University of California, writes as follows:

The results of an investigation on the absorption of sound in air, published by the author two years ago, indicated that, at least in the audible range of frequencies, the absorption coefficient (1) was from ten to twenty-five times greater than the coefficient calculated by classical theory, and (2) increased rather markedly as the humidity of the air decreased. The more recent work of Hopper and of Chrisler and Miller has led to similar results, although their experiments were not designed to determine the absolute values of the absorption coefficients. In addition, the work of Chrisler and Miller indicates that the absorption increases with a rise of temperature. In regard to the absorption coefficient of sound in air at 20° C., containing different amounts of water vapor for a tone of 6,000 cycles dying away in a six-foot chamber filled with air at 20° C., it will be seen that the rate of decay of sound was slowest for the most dry air; that it reached a maximum at a relative humidity of about 13 percent; and that it decreased again for higher humidities. Similar series of decay curves were obtained for frequencies of 500 to 11,000 cycles. In each case the rate of decay was slowest for the dry air; the rate of decay reached a maximum at a certain humidity, between 5 percent and 20 percent; and diminished at higher humidities.

In a recent communication to the writer, Professor Knudsen states:



Normally, oxygen molecules are of two types—those which are normal, or non-vibrating, and those which are in a state of vibration. In general, the number of vibrating or excited oxygen molecules is determined by the temperature of the gas. However, recent experiments which I have conducted on the absorption of sound in oxygen and water vapor indicate that collisions between oxygen molecules and water vapor molecules will result much more frequently in the production of a vibrating molecule than will similar collisions between two oxygen molecules . . . and further, *if water vapor will produce such a prominent effect upon the sound-absorbing properties of a gas, as oxygen, it is not at all impossible that the action of water vapor or oxygen molecules may result in an oxygen gas which has different chemical and physiological effects from oxygen which is free from water vapor, or from oxygen which contains a different percentage of water vapor.*

The following is a practical illustration of the effect of humidity on respiratory diseases. The writer, who lives in a city of over 100,000 population, on the seacoast, where the humidity average during the winter months is approximately 90 percent for the twenty-four hours, has conferred with pediatricists on the treatment of acute and chronic bronchitis and pneumonia (especially broncho-pneumonia). These specialists practice what they call the "Closed Window Treatment." This was found necessary because, while freezing is practically never known in this city, yet the combination of a temperature as low as 32 degrees F. is quite common in winter with a humidity, as stated, or close to 90 percent. To open windows in the sick room and permit a sick child, or adult, to inhale into a diseased lung this kind of air has proven very detrimental.

Some patients have, therefore, been sent to the desert. Others who have remained at home are treated by this closed window method, *i.e.*, the air is taken into the room and warmed to an appropriate temperature before being inhaled. By so doing, the humidity is lessened and the temperature is raised, without changing the constituent gases. Patients have recovered uniformly well under this treatment.

Also, the treatment of sinusitis during cold, humid weather in this seacoast region resembles this closed window therapy. For instance, the writer has found it very difficult to obtain good results on oil workers who are on night shifts of eight hours, exposed to cold, humid ocean breezes. The same applies to officers and men in the Marine Service, more especially the officers.

Consequently, instead of characterizing the sinus or chest patient as a neurotic, or rainbow chaser, the writer believes that through experimentation in the physicist's laboratory will be unravelled the reason why these cases become better oxygenated, develop better metabolism and have scantier secretions and freer drainage in certain

climates. To this should be added the therapeutic value of sunshine and winds, and even if climate does not amount to more than 10 per cent, in serious borderline cases 10 per cent often means the difference between success and failure.

## PART V

## RESEARCH (METEOROLOGICAL SURVEYS)

TABLE I.—METEOROLOGICAL

	Temp. (12 mo.)			Rela. Hum. (12 mo.)			Sunshine	
	Maximum Temp.	Minimum Temp.	Average Temp.	Maximum Hum.	Minimum Hum.	Average Hum.	Monthly Average Hrs. Sunshine	Percentage of Possible Hours of Sunshine
Group No. 1								
Vancouver* .....	..	..	49	..	..	80.41	152.75	..
Seattle† .....	80	37	58.5	..	..	80	152	..
Toronto .....	70.1	22.8	46.45	..	..	69	172.36	46
Winnipeg .....	70.09	-7.4	37.9	..	..	77	173.5	..
Group No. 2								
Miami Beach .....	83.8	62.8	73.3	78	74	76	249	67
Palm Beach .....	94	45	74.5	78	74	76	249	67
Group No. 3								
El Paso .....	81.7	44.8	63.25	54	27	40.5	..	90.70
Phoenix .....	103	38.5	70.75	56	28	42	37.04	84
Tucson .....	107	20	63.5	54.4	22.2	38.3	..	80

\* Vancouver (minimum relative humidity 71). These figures are not average for 12 months (maximum relative humidity 92) period.

† Seattle (no relative humidity record given but its proximity to Vancouver would lead us to assume that the relative humidity would compare with that of Vancouver.

*Table I.* Group 1 shows a group of cities which have low temperature, high humidity and few hours of sunshine. Group 2 shows two cities of Florida, which have high temperature, high humidity and a high percentage of hours of sunshine. Group 3 of this same table shows high temperature, low humidity and the greatest number of hours of sunshine.

*Table II.* It will be observed that from Group 1 the replies are practically all in the affirmative. From Group 2 and Group 3 the replies are all negative. In other words, out of twelve replies, eleven of the first group are in the affirmative; and out of the twelve replies in the second and third groups the entire twelve are negative.

TABLE II.—QUESTIONNAIRE TO DOCTORS

*Question:* Has your climate a tendency to produce chronic sinusitis which does not respond readily to treatment?

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Group No. 1	
Vancouver .....	Yes (1 reply)
Seattle .....	Yes (3 replies)
Portland .....	Yes (2 replies) No (1 reply)
Winnipeg .....	Yes (1 reply)
Toronto .....	Yes (4 replies)
Group No. 2	
Tampa .....	No (1 reply)
Palm Beach .....	No (1 reply)
El Paso .....	No (3 replies)
Group No. 3	
Phoenix .....	No (1 reply)
Tucson .....	No (1 reply)
Denver .....	No (3 replies)
Philadelphia .....	No (2 replies)

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A study of the two foregoing tables would indicate that climate is a definite factor in the etiology of sinusitis.

Following are excerpts from letters sent to the writer from doctors practicing in the abovementioned cities, which bear out the information revealed in the two preceding charts.

In reply to the question:

*Has your climate a tendency to produce chronic sinusitis which does not respond readily to treatment?*

*Vancouver, B. C.:* "Yes, in my opinion our coast climate does play a very important part in the causation of sinus infection and also in prolonging the treatment. Especially in our winter season we have quite a quantity of foggy weather and the atmosphere is generally damp. I notice that quite a number of sinus cases (mild cases of ethmoiditis) improve markedly when sent inland to the dryer climate. There is no doubt that most chronic cases of sinusitis require an operation, as there is always an anatomical abnormality present. I don't know how this compares with other locations, but having spent two and one-half years in New York, I must say I cannot see much difference between the two. These places, of course, are both foggy and situated near the water."

*Seattle:* "Temperature and humidity are probably factors in sinusitis, but I believe that sunshine is a very helpful agent for this winter (1933-34) we have had more sunshine than I ever remember, and far less sinusitis. In areas on this Puget Sound country where there is a great deal of wind, there seems to be less trouble than where we have little stirring up of the atmosphere."

*Florida:* "I do not think we see the percentage of chronic sinusitis that is seen elsewhere (that is in northern climate), due to the fact the common cold is not so prevalent, and clears up more rapidly in a mild climate. Of course we see a number of chronic sinuses, however the percentage is greater among those that have come from the North."

*Tucson:* "Would add that I have noticed, since being in this locality for fifteen years, that there has been only an extremely small number of patients, almost negligible, in my practice, residing here, who have been afflicted with sinusitis which has become chronic. However, I must say that I believe I note almost as much acute sinusitis as in more humid climates where I had formerly practiced."

*Denver:* "If our climate has any effect it is deterrent rather than productive of chronic sinusitis. . . . Lack of humidity and neglect of nose, with resultant dryness appears to be a factor in many cases. . . . Many cases improve to such an extent that they do not visit the doctor on arriving in Denver, and information to this effect is frequently gained incident to history of other conditions. . . . Improvement seems to be more marked for a time in individuals from other climates than in the case of Colorado natives."

It should be taken into consideration that the cities mentioned in the Pacific Southwest have a large tubercular population and, according to Hajek,<sup>17</sup> "it is truly remarkable, as shown by E. Frankel Wertheim, that nearly every third tuberculous body (evidently autopsies) was found with an inflammatory affection of the accessory sinuses. These inflammatory affections were not at all different from those occurring in the other diseases; they did not depend upon tuberculous infections since a true tuberculous infection of the mucous membrane of the accessory sinuses is rarely seen."

#### SUMMARY

We would conclude as follows:

1. That "just as one swallow does not make a summer," neither does one factor constitute an entire etiology. In this paper the writer deals with one cause only, namely, climate, and acknowledges the importance of all other factors.

2. That the nose is a factor in maintaining the water balance of the body, and the efficiency of its ciliary and other functions definitely depends upon changes in climate.

3. That the incidence of headcolds, while occurring about the same time of year over the entire continent and Europe, seems to be definitely related to changes in humidity and more especially to marked changes of temperature.

4. That a study of natural history suggests that the metabolic condition of all life, both wild and civilized, is affected by climatic changes.



5. That patients suffering from tuberculous disease of the upper respiratory tract appear to do better in certain climates.

6. That climate is a distinct factor in the etiology of diseases of the upper respiratory tract and, consequently, climate must be considered a factor in its treatment.

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# OBSERVATIONS ON THE LARYNX IN THE TUBERCULOUS\*

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The basis of this paper is the clinical study of the larynx in 452 cases of pulmonary tuberculosis with subjective complaints referable to the ear, nose or throat. Approximately 95 percent of these cases were considered ambulatory, and as a group on home or sanatorium treatment. The majority were chronic or subacute as to the lung stage.

The clinical invasion of the larynx by the tubercle bacillus is one of the most frequent complications of pulmonary tuberculosis that we, as laryngologists, are called upon to treat. In this study, particular attention is given to this complication.

## ETIOLOGY

*Frequency of Tuberculous Laryngitis.*—There is a wide variation in the statistics as to the frequency of tuberculous laryngitis. Ornstein<sup>1</sup> reports the frequency of tuberculous laryngitis as low as 4.4 percent, Looper and Schneider<sup>2</sup> 15.5 percent, Lockhard<sup>3</sup> an average of 34.5 percent in a series gathered from a large number of statistics, and Fetterolf<sup>4</sup> 83 percent, in a series of 100 autopsies. In my group, 87 or 19.2 percent had clinical tuberculous laryngitis.

These statistics are cited merely to show the wide discrepancy, and to emphasize the fact, that, to get an accurate picture we must analyze the lung stage and also the social condition of the patients. The literature reveals, in addition, that tuberculous laryngitis is found in much greater frequency in pathological examinations than in clinical studies. Spencer and Sumerill<sup>5</sup> have demonstrated tuberculous laryngitis in histopathologic studies antedating all clinical evidence of the disease.

## PREDISPOSING CAUSES

In this group of 452 cases, of all predisposing causes, only age, sex and pregnancy had any bearing on the development of this complication.

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\* Candidate's thesis.

*Age.*—Tuberculosis is primarily a disease of the young. In the largest percentage of cases these patients were under thirty, accounting for 41.5 percent of the group; 74.9 percent were below the age of forty years. In chronic and fibroid types, however, it is not uncommon to find active pulmonary tuberculosis in the aged. In this study, 6.4 percent of the patients were above fifty years, the oldest being sixty-six years.

Of the eighty-seven patients with tuberculous laryngitis, 31 percent were under thirty, 69 percent were above thirty. The youngest was nineteen years of age, the oldest fifty-nine years. From the comparison of these figures, it is noted that in the largest percentage of the pulmonary cases patients were under thirty years while the largest group of cases of tuberculous laryngitis was above thirty years.

Lockhard<sup>3</sup> states that there is an apparent immunity to tuberculous laryngitis in children. From my own and other statistics, there is apparently a relative immunity in the young. Dworetzky<sup>6</sup> did not find any cases in 100 children. Looper<sup>2</sup> reports only one case at ten years of age. On the other hand, Spencer<sup>7</sup> states that tuberculous laryngitis is rather frequent past forty-five. Wilkinson<sup>8</sup> reports five patients above fifty years, one at seventy-four, out of a group of forty-five cases. I believe that larynx invasion tends to come late in the course of the disease, irrespective of age, rather than that there is any specific or relative immunity in the young.

*Sex.*—Of the group, as a whole, 58.5 percent were males and 41.5 percent were females. Of the patients with tuberculous laryngitis 70 percent were males and 30 percent females, the relation being 2.33 times more frequent in the male. This would distinctly point to males being more susceptible than females and corresponds to statistics of Spencer,<sup>7</sup> Wilkinson,<sup>8</sup> Lockhard,<sup>3</sup> and others, although St. Clair Thomson<sup>9</sup> reports them approximately evenly divided. This male preponderance has been attributed to the more general use of tobacco and alcohol. Of all the males, however, history of the use of tobacco and alcohol was as frequent in the pulmonary cases as in the cases of laryngitis.

In four cases tuberculous laryngitis was precipitated by pregnancy. This is simply a result of the recognized deleterious effect of pregnancy in activating the general disease throughout the host, emphasized by Freudenthal.<sup>10</sup>

Other predisposing causes such as cold and warm climates, toxic agents, outdoor and indoor occupation, etc., were about equally divided in the cases of tuberculous and non-tuberculous laryngitis.

In this series, affections of the nose and throat were no more frequent in patients with tuberculosis of the larynx than in those where the disease was limited to the lung. These conditions are emphasized by many authors as important predisposing causes.

Local trauma is a factor, not noted, but which must be considered. Green<sup>11</sup> cites four patients, three of whom developed tuberculosis of the epiglottis following direct examination of the larynx, stressing the possibility that trauma of the speculum played a part in the spreading of the infection.

There are other predisposing factors, difficult to reduce to a statistical basis, in the incidence of tuberculous laryngitis such as the general resistance of the individual to tuberculous infection. This resistance may be hereditary, racial or acquired in early childhood and can occasionally be estimated by the blood sedimentation time, shift to the left, and pulmonary response to treatment. There is, in addition, the local immunity spoken of by Fenton.<sup>12</sup>

The exciting cause of tuberculous laryngitis is the invasion of the larynx by the tubercle bacillus. The modern conception is that it is always secondary to tuberculosis elsewhere, and this is supported by clinical and pathological evidence of most authorities. When we consider that most individuals contact the tubercle bacillus in infancy or early childhood, the possibility of primary invasion of the larynx is at least remote. Spencer<sup>5</sup> states he has never seen a primary case. Lockhart,<sup>3</sup> Arrowsmith<sup>13</sup> and others also question its existence, while Wood<sup>14</sup> believes it does occur, but is extremely rare. Terry,<sup>15</sup> Newhart<sup>16</sup> and others have found tuberculosis in from 5 to 42 percent of tonsils showing no clinical signs of the disease. Tuberculosis is found postmortem in a large percentage of tracheobronchial glands. Therefore, the primary focus may at times be latent and extremely difficult to demonstrate clinically. All of my cases were secondary to clinical pulmonary tuberculosis.

#### ROUTE AND MODE OF INFECTION

Most observers believe that infection of the larynx comes through contact with bacterial laden sputum passing over and frequently resting, for a long period of time, in the larynx. Other prominent workers in this field feel that infection is usually through the lymphatics, citing the fact that pathologically, the tubercles are subepithelial and none occur in the epithelium. Supporting the former theory, however, is the fact that tubercle bacilli can penetrate normal mucous membrane without leaving any surface lesion, and that tuberculous lesions are most frequently formed in the posterior portion of the



larynx, where the sputum from an anatomic standpoint is prone to lodge.

Personally, I feel the sputum infection route is the best supported clinically, and the most frequent route. In my series of cases the sputum was positive approximately as frequently in the cases of non-tuberculous laryngitis as in those of tuberculous laryngitis. The sputum, in 82 percent of the cases, was positive at some time in the course of the disease. In Wilkinson's<sup>8</sup> series of forty-five far advanced and neglected cases of tuberculous laryngitis, the sputum was negative in 22 percent of the cases. Dobromylski<sup>17</sup> found 9 percent of tuberculous laryngitis with a bacillar sputum, suggesting that these cases are hemotogenic. Green<sup>11</sup> states that the mode of infection is of scientific interest only, and has no bearing on the course or prognosis of the disease.

The low incidence of tuberculous laryngitis, in active pulmonary cases with positive sputum, is noteworthy. It suggests a high degree of specific tissue resistance to clinical invasion of the tubercle bacillus in the upper respiratory tract. Clinical tuberculosis of the nose, sinuses, tonsils and pharynx, tongue and buccal cavity, is comparatively rare, except as terminal spreading lesions. Terry<sup>15</sup> found only one case of tuberculosis of the nose, none of the sinuses, ten of the tongue, in a large series, over a period of three years. Havens<sup>18</sup> was able to report five cases in the nose, and collected fifteen in the American literature. In my series there was none in the nose or sinuses, one of the tongue, three of the tonsil, one of the pharynx, all associated with tuberculous laryngitis. There were, however, twenty-five cases of tuberculous otitis media, two of tuberculous mastoiditis in nontuberculous laryngitis cases, four tuberculous glands of the neck, one of which had tuberculous laryngitis.

On the other hand, tuberculous patients possess no immunity to other upper respiratory infections, in fact, from the general lowered resistance they seem more susceptible to common colds, sinusitis, laryngitis, etc. In the group of cases where tuberculosis was limited to the lungs, there were sixty-five cases of acute or chronic laryngitis, sixty-eight cases of sinusitis, fifteen cases of pharyngitis, fifty-nine cases of acute or chronic otitis media, sixty-seven cases of infected tonsils, twenty-one cases of allergy, twenty-two cases of deflected septums, thirty-six cases of atrophic rhinitis. I want to emphasize that these affections were of importance symptomatically, and consultation for their relief requested by the lung specialist. In nineteen patients, tonsillectomy was performed under local anesthesia. The indications were definite and clear and operation advised only

after a period of observation, during which the general condition of the patient failed to respond to conservative treatment. One patient was given artificial pneumothorax forty-eight hours previous to operation, as a preliminary preparation. The indication was from the lung standpoint. Section of four of the tonsils from these cases failed to reveal any tuberculous infection. All healed promptly, improved generally and none developed tuberculous lesions in the upper respiratory tract, over a period of two years.

The relative infrequency of tuberculosis in the upper respiratory tract is remarkable when we consider that the tubercle bacillus gains entrance to the host, either by inhalation or injection, and must pass over or through these upper respiratory structures, certainly more subject to exposure and trauma than the lungs or tracheobronchial glands, where tuberculosis is found in much greater frequency.

There is one factor that I feel plays an important part in the development of tuberculous laryngitis. Pottinger<sup>19</sup> has called attention to the parasympathetic trophic reflex, in which the afferent impulses course in the pulmonary branches of the vagus and the efferent fibres course in the laryngeal branch. Irritation of these afferent fibres by inflamed pulmonary tissue produces trophic changes and atrophy in the larynx. Atrophy was noted in 9 percent of the tuberculous laryngitis cases. This disturbance Pottinger believes lowers local tissue resistance in the larynx and offers a reasonable explanation for the fact that tuberculous laryngitis is practically always secondary to pulmonary tuberculosis, and if the lesions are unilateral they usually occur on the side of the pulmonary disease.

*Pathology.*—The pathology of tuberculous laryngitis is essentially that of tuberculosis elsewhere, modified by the anatomic structure and location in the larynx. Microscopically, it is a chronic granuloma, an inflammatory or allergic response to the tubercle toxin. There are round cell infiltration and epithelioid cells about the bacilli and occasional giant cells of Langhans. There is frequently coagulation or caseous necrosis at the center of the tubercle which is avascular and is located in the subepithelial layer, but may necrose to the surface and involve the epithelium, resulting in ulceration either by pressure or round cell infiltration.

Grossly, in the larynx the pathology is extremely protean, usually more than one type of lesion occurring in the same larynx. Of my cases only three had solitary lesions. It may simulate almost any of the numerous diseases of the larynx and is, in fact, frequently complicated by secondary or coexisting nontuberculous disease. Grossly, we see congestion, anemia or extreme pallor, atrophy, infiltration,

edema, tubercle, tuberculoma, ulceration, perichondritis and chondritis. Types of lesions noted in this series in order of their frequency were as follows: Infiltration or edema, 96.6 percent; asymmetrical hyperemia, 65.6 percent; extreme pallor or anemia, 20.4 percent; ulceration, 20 percent; tubercle, 9.1 percent; atrophy, 9 percent. Various stages of paresis and paralysis of the cords are generally manifestations of pressure and traction on the recurrent laryngeal and therefore not considered as true laryngeal disease.

#### LOCALIZATION OF LESIONS

In 76 percent, lesions were bilateral, in 24 percent, unilateral. Asymmetry of lesions, with the possible exception of cases where disease was limited to the epiglottis, occurred in all the cases. The order of their frequency was: interarytenoid space, 46 percent; cords, 43 percent; epiglottis, 36 percent; arytenoids, 30 percent; ventricles, 24 percent; subglottic area, 5 percent.

*Symptoms.*—The symptoms of tuberculous laryngitis depend on the type of lesion present (laryngeal stage), and on its localization. The principal subjective symptom of intrinsic localization is voice change. This may vary from a slight huskiness to vocal fatigue, from dysphonia to complete aphonia. Parasthesias, tickling, foreign body sensation and dryness are frequent complaints, occasionally reflex cough. My experience is that the diagnostic value of these symptoms is questionable as they are found frequently in the cases of non-tuberculous laryngitis. It is noteworthy that pain, and in fact all subjective symptoms, may be entirely lacking as Solenberger<sup>20</sup> points out, even in disintegrating types with extrinsic involvement. However, where the disease is extrinsic, pain is usually one of the most distressing complaints. This is not constant, but expressed as sharp and knife-like, excruciating on swallowing or talking, and frequently referred to the ear. Dyspnea occasionally may occur due to narrowing of the glottis by infiltration, edema and tuberculoma. However, this symptom is also due to narrowing from cord paralysis, and at times, "air hunger" from extensive lung disease or bronchial stenosis.

Because of the extremely protean character of tuberculous laryngitis, the objective findings are varied and there is really no typical early picture. General congestion of the larynx was noted earlier and more frequently than extreme pallor, but while these findings are sometimes spoken of as pathognomonic, unless they occur asymmetrically or in conjunction with infiltration, one of the earliest

pathological processes, they cannot be considered indicative of clinical tuberculous laryngitis.

There are, however, characteristic findings in various portions of the larynx. In this series, the posterior wall was the most frequently involved. The earliest sign noted was an irregular thickening of the mucous membrane or wrinkling, best seen on phonation. As this process advances, this thickening takes on the appearance of irregular digitations, and as these enlarge and coalesce a thick pad or mass forms, sticking out between the cords. As the tubercles enlarge and come to the surface or break down, ulceration occurs. The ulcers are at first superficial and irregular, the base being covered with yellowish exudate or sputum. Later as necrosis proceeds, they spread, involving the deeper structures, and the edges and base may contain granulations.

Next in frequency was vocal cord involvement. The earliest evidence is a rounding, usually in the posterior third, giving the cord a spindle shape, and vessels may be seen coursing the surface. Superficial ulceration may take place giving the cord a "mouse-eaten" appearance.

*Epiglottis.*—In the epiglottis, the first sign was a thickening of the free edge. The epiglottis was injected, firm, sometimes rather granular or warty in appearance. This swelling may increase with edema involving the entire structure, giving it the typical turban shape. Ulcers usually appear on the free margin, are rather cleancut, exposing the edge of the cartilage. This may spread until the entire epiglottis is destroyed. This ulcerative process is also seen at times in the far advanced cases as a disintegrating type, where there is widespread destruction with only slight infiltration and tissue reaction.

*Arytenoids.*—The first sign noted is a small, rounded, pale, edematous swelling assuming a pear shape as it increases. This may reach a tremendous size, obstructing a clear view of the cords, and at times causing difficulty in swallowing and closure of the larynx, and rarely, obstruction to breathing.

*Ventricles.*—Here infiltration produces a rounded mass which overhangs the cords and may simulate prolapse of the ventricle. Subglottic involvement occurred in only 5 percent of the cases and consisted of infiltration, causing more or less obstruction to breathing.

*Diagnosis.*—In the vast majority of cases, diagnosis of tuberculous laryngitis can be made by what one sees in the larynx. This may necessitate repeated observations and direct examinations. The confirmatory aid of the lung specialist, laboratory, sputum, Wassermann,



x-ray and at times, biopsy, completes the diagnosis. Biopsy, however, is seldom necessary or advisable, except to rule out the possibility of malignancy. We must not be too willing to conclude all larynx lesions are tuberculous. Green<sup>21</sup> and others have shown a large number of nontuberculous lesions in the tuberculous.

X-ray of the larynx in recent years has received enthusiastic acclaim in diagnosis by Woods<sup>22</sup> and Jackson,<sup>23</sup> due to the work of Thost and Wotzilka in Germany, and of Pancoast in this country.

#### DIFFERENTIAL DIAGNOSIS

Chronic catarrhal laryngitis must be ruled out because this is so frequently found in the larynx of the tuberculous. It was noted in sixty-five or 14 percent of all the cases examined. There is a general redness of the larynx—all the mucous membrane tends to be equally affected, in contradistinction to asymmetrical lesions usual in tuberculosis.

Syphilis is prone to attack the anterior portion of the larynx, the injection is more of a brick red, ulcers are sharp and punched out. The lesions tend to be more extensive, destroying identity of structures. Wassermann and therapeutic tests help clarify the picture.

Carcinoma may resemble tuberculous laryngitis. It is generally a more localized lesion, smaller and usually unilateral. Personally, when doubt exists, I have no hesitancy in doing a biopsy, provided the patient is prepared and physically able to proceed with radical treatment. If the condition proves to be tuberculous, the raw edges are cauterized. We must, however, bear in mind Lynch's<sup>24</sup> warning, "I have made the mistake a number of times of believing the report of one microscopic section." The two conditions may co-exist, and Tucker<sup>25</sup> reported a case with typical findings of both, in the same section.

Laryngitis sicca, especially when the cords are injected, hemorrhagic, and have crusts, may have to be differentiated. The lesions are tenacious crusts, and the membranes are thin, dry and glazed. As a rule, ulcers are not present, and usually atrophic rhinitis is present. Rarely pachyderma laryngis, scleroma, lupus and leprosy must be ruled out. Hysterical aphonia was not noted in this series but its occurrence, especially in nervous individuals, must not be overlooked.

*Prognosis.*—Prognosis depends on so many variable factors, that statistics are of little value in making a definite decision. The occurrence of tuberculous laryngitis materially and unfavorably affects the general prognosis as to life, in fact, it frequently determines the

ultimate outcome. All the older authors considered the prognosis hopeless and treatment futile.

Early diagnosis, and, more recently, our advances in surgery in the treatment of both pulmonary and laryngeal tuberculosis, have improved the prognosis of this serious complication. Each year has seen various authors better the prognosis. Spencer<sup>26</sup> states that seventy to eighty percent are curable, if the diagnosis is made early, and Woods<sup>27</sup> states that as high as 90 percent can be cured.

In my series fifteen lesions or 17.2 percent are considered healed; forty-seven or 54 percent improved or quiescent; nine or 19.2 percent unimproved and sixteen or 18.4 percent patients have died. Of the latter, two died directly from tuberculous meningitis, one from Addison's disease, one from persistent hiccough which could not be controlled even by bilateral phrenicotomy, the others from their pulmonary disease. These cases have been followed over a period varying from a few months to five years. My experience is that the favorable prognosis applies only to the healing of the local tuberculous lesion in the larynx. St. Clair Thomson<sup>28</sup> brings out the significant point that, out of 2,541 patients discharged from King Edward's Sanatorium, the proportion of those alive between one to ten years later, was twice as high among those with sound larynges, as among those who had had tuberculous laryngitis.

*Treatment.*—In the treatment of tuberculous laryngitis, we must remember we are treating a secondary infection from a primary focus in the lung. Therefore, attention to the lungs should receive primary consideration. However, as this secondary invasion of the larynx frequently determines the final issue, it is important to attack it vigorously, along with the treatment of the primary focus.

Prophylaxis should be practiced by routine and repeated examination of the larynx of all tuberculous patients, when their general condition permits, and there should be close cooperation with the lung specialist in the general management of the patient. On the appearance of early or suspicious symptoms, absolute rest of the voice is advisable. This alone has been sufficient to prevent or cure an early case. Whispering may be as harmful as talking, as Carmody<sup>29</sup> has pointed out. Attention should be given to nose and throat hygiene, and a simple, generous diet, rich in vitamins A and D is important. Where pain and dysphagia are present, it is essential to give relief and to see that the patient receives sufficient food and fluids to maintain the general condition. Many of these patients actually fear to eat on account of the acute pain on swallowing. It has been noted that patients have individual idiosyncrasies in this regard. One

may be able to take liquids freely and have trouble with solid or semi-solid food. In many cases the reverse is true. Many can swallow acid or tart food, and others only sweetened foods. This must be worked out in the individual case. Then again, swallowing is sometimes made easier by hanging the head over the edge of the bed so that the head is lower than the body and taking food through a straw (the Wolfenden position). Others are helped by tipping the head backward, similar to the position used in feeding intubated patients.

Many local treatments are advocated for palliative relief, such as spraying with cocaine, euphagen, butyn, etc. Curative use of formalin, menthol, chaulmoogra oil, lactic acid, carbon dioxide snow, tuberculin, etc., all have their advocates.

In my hands, in early cases, general care, absolute rest of voice and any oily application to the membrane, has given the best results. Lukens<sup>30</sup> has reported favorable results with chaulmoogra oil. It is my opinion, that in early cases there is much more danger of over treatment than under treatment. Indiscriminate swabbing of the larynx, especially with silver solutions, which produces transudation, may cause abrasions opening new avenues to secondary infection. I have found alcohol injection of the superior laryngeal nerve a valuable temporary aid in combating pain. This is a simple procedure well tolerated by even acutely ill patients, and it may be administered at the bedside. Others prefer section of the nerve. Reflection of sunlight into the larynx by means of Verba mirrors is a valuable adjunct to treatment, and some authors report excellent results. However, many patients do not acquire skill enough to direct the rays properly, and those who do, tend to overtreat themselves, at times resulting in sunburn of the larynx. In selected cases, however, under close supervision, it is of value.

Wessely<sup>31</sup> in Vienna, Miller,<sup>32</sup> Katz<sup>33</sup> and other authors in this country, have reported favorable results by the use of Quartz and other types of ultraviolet lamps, directed into the larynx largely under direct methods. Most of the cases I have seen do not tolerate direct laryngoscopy well, especially when this has to be repeatedly done and there is also danger of trauma and extension of the tuberculous processes. X-ray and radium have been advocated, but their use is not without danger.

Galvanocautery has been the one most valuable agent in treatment. This is carried out under the indirect method, in all but exceptional cases. It should be used with caution and on selected cases. I have used it on the cases of swollen and granular epiglottitis, on

granulations, interarytenoid digitations, pale gray edemas of the arytenoids and ulcerations. Where other methods have failed to give relief from pain, I have even used it in active and acute cases. Local anesthesia is used and there is no special preparation of the patient. The amount of cauterization and frequency of use and type of cautery depends entirely on the general condition of the patient, and the type of lesions present. Ulcers are superficially cauterized. The masses are punctured from one to ten times. This may be done at one sitting or repeated from a few days to several weeks apart. The main point is not to exhaust the patient at any one sitting. The most striking feature of cautery puncture is the relatively slight local reaction, and secondly, the almost constant alleviation of pain. I have had patients ask for repeated cauterizations because of the relief of pain. It seems to me that physiologically, the cautery puncture is a correct procedure. We know that tuberculosis heals by fibrosis. Burns produce scars and frequently excessive scar, which aids in the healing. The cautery destroys bacteria actually reached, and pasteurizes neighboring areas. Green<sup>34</sup> has shown that the cautery produces an inflammatory reaction, with the development of fibroblasts and new blood vessels in the surrounding tissues. The beneficial effect of the cautery, therefore, extends considerably beyond the local point of application. No attempt is made to destroy all the diseased tissue by the cautery, but only to stimulate this healing process.

Another procedure I have found valuable is rather out of the laryngological field. This is lung collapse, either by phrenic evulsion, pneumothorax, or thorocoplasty. Dworetzky<sup>35</sup> reports a remarkable series of cases in which the frequency of tuberculous laryngitis was lowered to about .3 of 1 percent and many cases of tuberculous laryngitis were cured by this procedure. Therefore, if any of the pathognomonic early signs of tuberculous laryngitis are noted, the laryngologist, in cooperation with the lung specialist, should recommend lung collapse, when consistent with the pulmonary stage. Many of my cases had lung collapse, but indications were primarily from the lung standpoint.

I have had three cases of tracheotomy performed for acute obstruction to breathing; one with bilateral abductor paralysis and two with subglottic infiltration and massive involvement of the larynx. In every case, there was improvement noted in the laryngeal disease and also in the chest findings. The paralysis, however, was not benefited in any way. Tracheotomy also permitted the radical use of the galvanocautery, which was used for the relief of pain, in two cases. Tracheotomy is only indicated for marked laryngeal obstruction, but when



necessary, secondarily, we can expect improvement to the laryngeal disease.

### SUMMARY

The larynx was studied from the clinical standpoint in 452 cases of pulmonary tuberculosis, with subjective symptoms referable to the ear, nose or throat. Tuberculous laryngitis was present in 19.2 percent of the cases. This is somewhat higher than would be expected in the mere routine examination of the larynx in the tuberculous, without subjective complaints. Lung stage, social conditions, and clinical or pathological diagnosis must be considered in analyzing the statistics.

In this group, only age, sex and pregnancy had any bearing on the development of tuberculous laryngitis. Affections of the nose and throat were no more frequent in patients with tuberculous laryngitis, than in those where the disease was limited to the lungs. Tuberculous infection in the larynx usually comes from contact with bacteria laden sputum, although it is probable that some cases, with a bacillar sputum, are lymphatic in origin. There is a strong local resistance noted to clinical invasion of the tubercle bacillus in the upper respiratory tract, but no such immunity is noted for other infections. The parasympathetic trophic reflex plays a part in breaking down local resistance, and offers a reasonable explanation for the fact that tuberculous laryngitis is secondary to pulmonary disease. The pathology is essentially that of tuberculosis elsewhere, is extremely protean, more than one type of lesion occurring in the same larynx and frequently complicating secondary nontuberculous lesions. Subjective symptoms are of little diagnostic value and the diagnosis is based on the gross findings in the larynx, with the confirmatory finding in the chest, laboratory and x-ray. Prognosis as to healing of the laryngeal lesion in early cases, is good, but guarded as to ultimate recovery. Early treatment is based on cooperation with the lung specialist in the general care, on absolute rest of voice, lung collapse therapy in suitable cases and on avoiding overtreatment locally. Occasionally, reflected sunlight in supervised cases is of value. In progressive cases, radical and repeated use of the galvanocautery is of great worth, care always being taken not to exhaust the patient at any one sitting. Time and temporary relief may be gained by superior laryngeal nerve block.

The actual cautery is well recognized now as one of our most valuable aids. I feel its use has been too frequently reserved for far advanced and more or less hopeless cases. Early use is urged,

especially in cases which do not promptly respond to conservative treatment.

Probably in no other field of medicine, with no known specific remedy, has such notable progress been made in combating the incidence and course of such a widespread disease, as in pulmonary tuberculosis. This is basically the primary focus of laryngeal tuberculosis. Intelligent cooperation between the phthisician and laryngologist will result in equally striking results in the prevention, early diagnosis and successful treatment of this serious complication.

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# THE EFFECT OF PHYSICAL AGENCIES UPON THE TEMPERATURE OF THE NASAL SINUSES\*

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For a number of years, the writer has been interested in the relative value of various forms of treatment used in the nonsurgical care of acute and subacute infections of the paranasal sinuses. The part played by heat and cold was particularly interesting, as a marked difference of opinion existed among physicians as to just what happened when the various methods were employed. It seemed that it would be a comparatively simple problem to conduct some experiments which might help to determine the effect of the several methods.

A search of the literature failed to reveal any reference to work of exactly this nature. Quite a bit had been done, however, on the penetration of heat into various more solid structures, such as the orbit.

Moncreiff, Coulter and Holmquist<sup>1</sup> found that the penetration of heat into the orbit was greater with infra red lamp than with an electric heating pad. They felt that the ability of the skin to tolerate heat largely determined the amount which penetrated beyond it. Two degrees Centigrade more was tolerated by the skin, when infra red was used, than with the electric heating pad.

Cone, A. J.<sup>2</sup> made some temperature readings of the mucous membranes of the nasal passages. He found these temperatures higher in patients with infection in the sinuses and in those whose basal metabolism was above normal. The temperatures were lower where metabolism rates were low and where allergy existed.

Krukower<sup>3</sup> found that the body temperature influenced the temperature of the sinuses to a slight degree, while the temperature of the outside air had very little, if any, effect. He also found that the temperature of these membranes was increased in acute and subacute infections, but was not altered in chronic infections.

Lewis and Pickering<sup>4</sup> found that raising the body temperature caused a vasodilatation of the blood vessels of the extremities, when these were exposed to the outside temperature while the remainder of the body was being warmed in a cabinet. They felt that this was

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\* Candidate's thesis.



the means the body employed to dissipate abnormally high temperatures in isolated portions of the body.

### GENERAL SCOPE OF THE EXPERIMENTS

Plans were made to record the changes in the temperature of the sinuses when cold compresses, ice bag, hot compresses, hot water bottle, or a heating lamp was applied over the sinuses, or when the whole head was heated by the high frequency oscillating current, the so-called radio heat.

### APPARATUS USED TO APPLY HEAT AND COLD

Small bath towels wrung out of ice water were used for cold compresses. The ordinary commercial rubber bag filled with finely cracked ice was used for ice bag. Small bath towels wrung out of boiling water and applied to the face as soon as the skin could tolerate them were used as hot compresses. The electric heating pad used was a rubber covered heating unit, operating at 115 volts and .1 ampere, with a cloth outside cover. The lamp had a 260-watt bulb, with carbon filament. The reflector was polished metal and was  $7\frac{3}{4}$  inches in diameter, and the lamp was set so that the rays would strike the cheek at right angles at a distance of 18 inches from the bulb to the skin.

The high frequency oscillator used in these experiments was constructed on the same principle as a short wave radio transmitter, with the exception that the energy is concentrated between two condenser plates instead of being directed from an aerial. The heater was designed by the General Electric Company, and consists of a vacuum tube oscillator and rectifier that supplies the high voltage for the oscillator. The high frequency oscillator is composed of two 75-watt radiotrons operating at a frequency of from 9,000 to 12,000 kilocycles. An air cooled transformer, having a 4,500 volt secondary and feeding a full wave rectifier, forms the 2,000 volt direct current plate supply for the oscillator. The patient's head was placed between these plates, almost filling the space of 16 inches between them. Figure 1 shows the apparatus set up. Figure 2 shows details of the hook-up.

### APPARATUS USED TO RECORD TEMPERATURE CHANGES

To record the temperature changes within the sinuses, a thermopile of four junctions, each consisting of a strand of No. 30 constantan and No. 30 iron wire, was used. Each junction was electrically welded, and copper wires connected the terminals of the pile with the

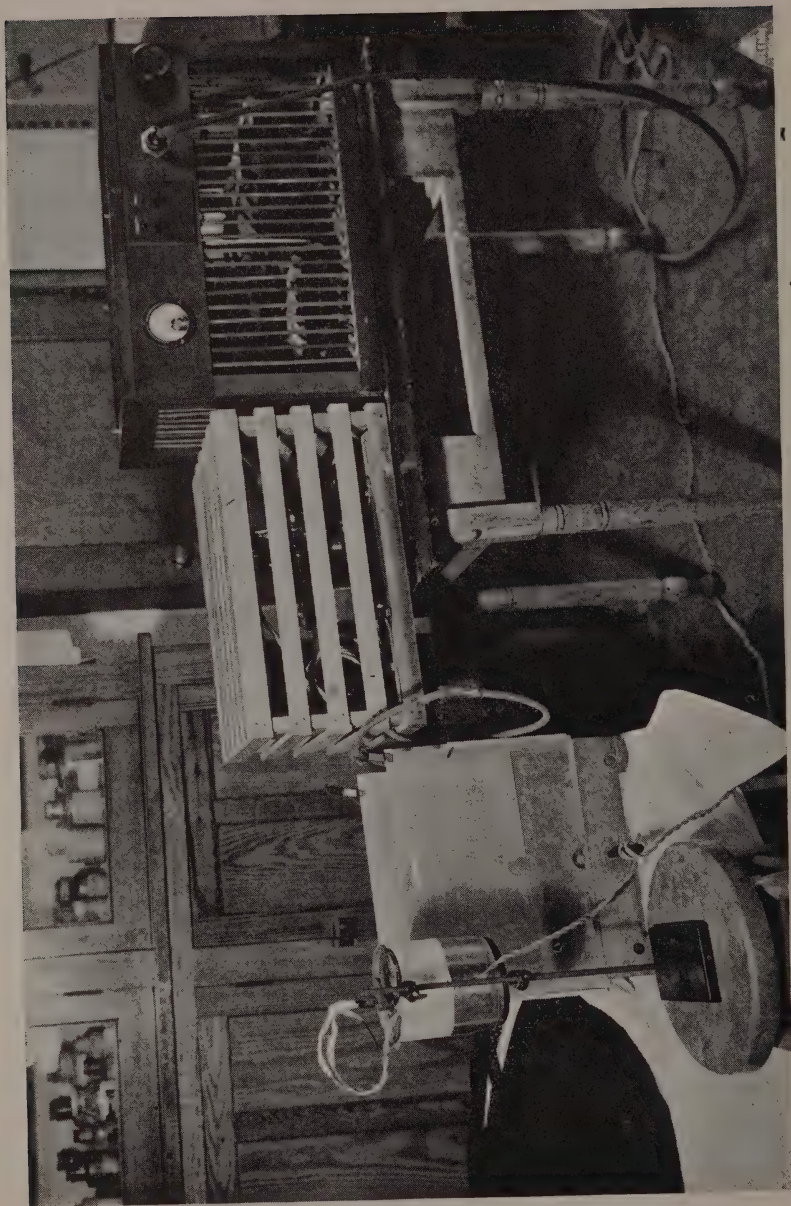


FIG. 1. Apparatus set up. With patient's head between condenser plates. Cold junctions container on stool.

registering galvanometer. The resistance of the whole assembly was found to be about 21 ohms. The individual wires were given three coats of flexible waterproof enamel and were made into a single cable by tying them together with fine silk thread. The cable was given several additional coats of the same enamel and was rubbed down to smooth out the rough spots.

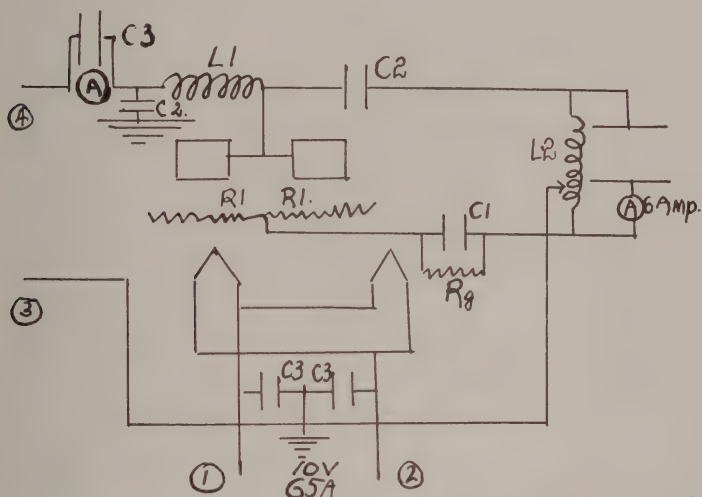


Fig. 11 Wiring Diagram 30 Meter Oscillator 2 UX852 Tubes  
 A1 6 Amp. Ther. Couple Do6 C1 .002 Condenser UC1014 or 1874-K  
 A2 0.5 Amp. Meter DO.4 C2 UC 2224.  
 L1 Choke Coil C3 Faradon Model T Cond. .01 Cap  
 L2 8 Turns cu.  $\frac{1}{4}$ " x  $\frac{5}{16}$ " Rg Large Blue Sticks 10,000 Ohms.

FIG. 2. Detail of high frequency oscillator wiring diagram.

The cold junctions were immersed in about 5 c.c. of 95 percent alcohol contained in a small tube which was in turn inserted through a rubber stopper. This was fitted to the top of a thermos flask filled with finely crushed ice. The flask was wrapped in vegetable wool and placed in a metal can with only the neck of the flask protruding.

The thermoelectric voltage generated by this sensitive thermopile was found to be so great that the mirror galvanometer had its readings pushed entirely off the scale. It was therefore necessary to bring the readings back on the scale by the application of an opposing voltage in the thermopile circuit. This current was adjusted by interposing several variable resistance units. Figure 3 shows the recording apparatus. Figure 4 shows the hook-up.



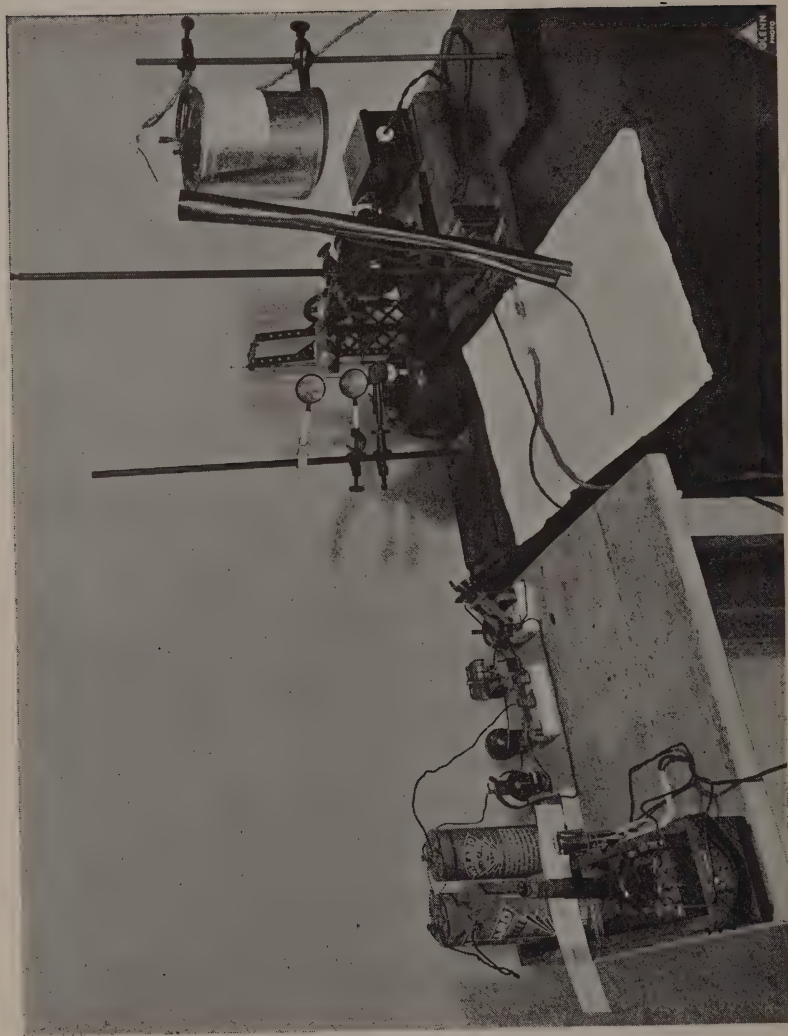


FIG. 3. Recording apparatus. On table, to left, galvanometer, variable resistance units, and switches closing circuit. On table, to right, container for cold junctions, showing thermopile with leather insulator. In background, thermometer for calibrations, lenses for reading same, and mechanical stirrer, motor and resistance units.



The thermopile was calibrated by comparison with a thermometer graduated to  $0.2^{\circ}$  F. The thermopile tip was loosely fastened to the bulb of the thermometer which was immersed in water kept in constant motion by an electric stirring apparatus. Several hundred readings were made at intervals of  $0.2^{\circ}$  F. from  $106^{\circ}$  to  $96^{\circ}$  F. It was found that  $1^{\circ}$  F. averaged about 1.87 large divisions on the galvanometer. As each large division on the galvanometer is graduated into

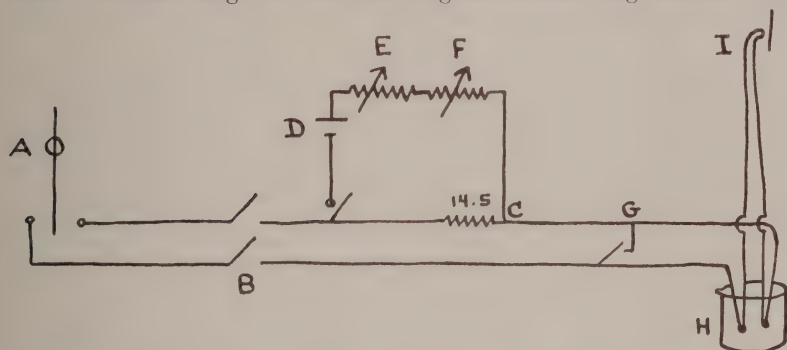


FIG. 4. Wiring diagram of recording apparatus. A, galvanometer; B, main cut-out switch; C, shunt resistor, 14.5 ohms; D, the battery; E and F, variable resistors; G, switch for short-circuiting thermopile; H, cold junction assembly; I, the working tip (only two junctions are shown for simplicity).

tenths, the instrument could be read directly to about one-nineteenth of a degree Fahrenheit. The original aim in the design of the apparatus was an accuracy of  $.1^{\circ}$  F. The surplus sensitivity merely reduced the average error. A series of readings by different observers indicated that the galvanometer variations, per degree of temperature change, were practically the same within the limits to which the thermometer could be read, and that the temperatures could be read directly from the galvanometer as equal divisions of the deflection between two end temperatures without calibration for each intermediate interval. Before and after each experiment, the thermopile was checked in water of known temperatures to insure accuracy.

In several of the experiments where the stem of the thermopile cable was directly exposed to heat or cold from the apparatus it was protected by several layers of thin leather. Control tests also proved that heat, applied to the stem of the thermopile, had absolutely no effect upon the reading of the instrument.

When the high frequency oscillator was used to heat the subject's head, it was found that currents induced in the galvanometer circuit seriously interfered with the temperature readings if the oscillator was close to the registering apparatus. It was necessary to move the oscillator about fifty feet away before this trouble was overcome.

When this apparatus was used in the experiments, the thermopile was placed in the patient's sinus and allowed to remain there until the readings became stabilized. It was then removed, while the oscillator was in operation, and was quickly replaced after the current was shut off.

When the tip of the thermopile did not completely fill the opening into the various sinuses the remaining opening was completely closed by a cotton packing.

### TYPE OF PATIENTS USED

When the work was first planned it was intended that animals should be used. Later it was felt that the results might have a more practical value if the experiments were done upon human sinuses.

Three patients were used in the experiments to be described:

*Patient "A."*—Male, age eighteen, weight about 150 pounds, bacterial asthma of fourteen years' duration. Had had septum resected, antra ventilated, a complete ethmoidectomy, and the anterior wall of the sphenoid had been partially removed. The nose was clean, but the membranes were thickened.

*Patient "B."*—Female, age twenty-two, weight about 125 pounds, asthma of ten years' duration, had had same operations as Patient A. Nost was clean, and membranes slightly thickened.

*Patient "C."*—Male, age fifty-two, weight about 150 pounds, had had asthma about twelve years, same operations as Patients A and B with the exception of no sphenoid opening.

None of these patients had any evidence of acute infection in their sinuses when the tests were made. Each was the subject for numerous experiments over a period of two years.

### EXPERIMENTAL DATA

#### *Experiment 1—Patient A*

The thermopile was placed in the right nostril. The temperature registered 92.6° F. It was then placed in the left nostril and the temperature registered 90° F. Upon inspiration the temperature was about 2° F. lower than upon expiration. When the thermopile was removed the right nostril appeared to be somewhat obstructed as compared to the left nostril.

#### *Experiment 2—Ice Bag—Patient A*

The thermopile was placed in the right antrum. When the galvanometer readings became stabilized, in fifteen minutes, an ice bag was placed over the right antral region of the face. This was left in place for one hour. No definite change in the readings was recorded.

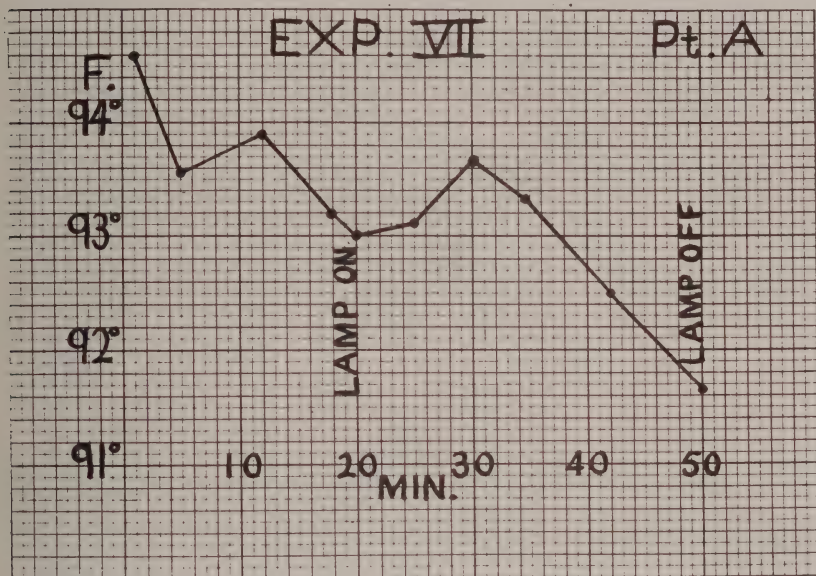
#### *Experiment 3—Cold Compresses—Patient A*

The thermopile was placed in the left antrum. When the galvanometer readings became stabilized, in fifteen minutes, cold compresses, wrung out of

ice water, were placed over the left antral region, and were changed as they warmed, for a period of one hour. No definite changes in the readings were recorded.

*Experiment 4—Hot Water Bottle—Patient A*

The thermopile was placed in the left antrum. When the galvanometer readings became stabilized, after fifteen minutes, a hot water bottle was placed over the left antral region. This remained in place for one hour. No definite changes in the readings were recorded.



*Experiment 5—Electric Heating Pad—Patient A*

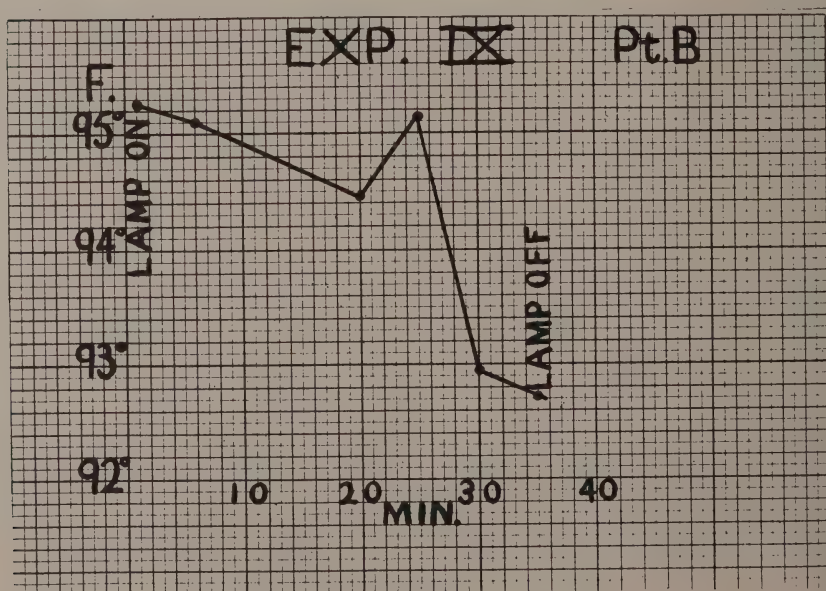
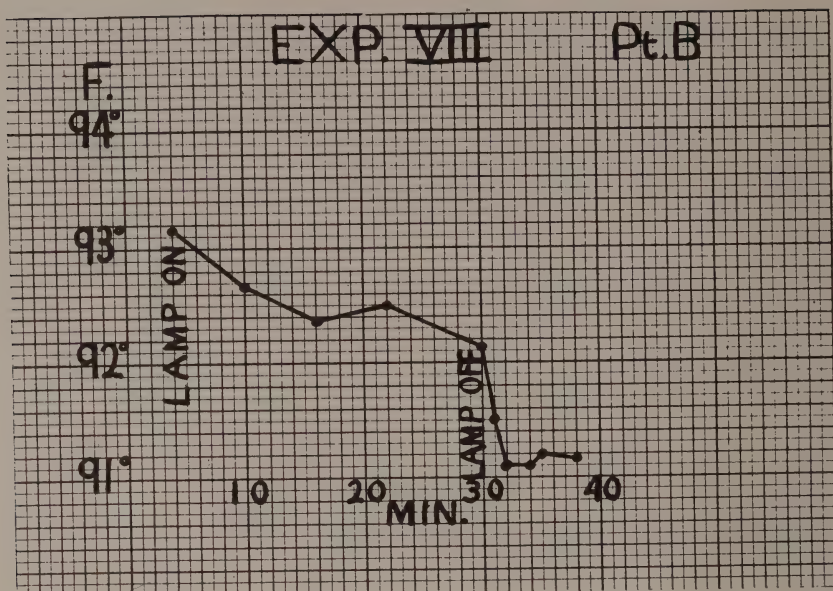
The thermopile was placed in the right antrum. When the galvanometer readings became stabilized, after fifteen minutes, the electric heating pad was placed over the right antral region and the switch was turned to the high position. The pad remained in place for one hour. No definite changes in the readings were recorded.

*Experiment 6—Hot Compresses—Patient A*

The thermopile was placed in the left antrum. After the readings were stabilized, in fifteen minutes, compresses, wrung out of boiling water, were applied to the left antral region as soon as the skin would tolerate them. These were changed every five minutes, as they cooled, for a period of one hour. No definite temperature changes were recorded.

*Experiment 7—Radiant Heat—Patient A*

The thermopile was placed in the right antrum. The antral window was not closed with cotton. After the readings were stabilized, in fifteen minutes,





the lamp was put into the position described before. The lead and the exposed portion of the thermopile were protected by several layers of leather.

After the lamp had been turned on for twenty-two minutes, the antral temperature began to drop. At the end of one-half hour the temperature had dropped 1.4° F.

About twelve minutes after the lamp was on, the patient noted an increase of drainage from the nose. It appeared to be of a serous character when it was expelled.

#### *Experiment 8—Radiant Heat—Patient B*

The thermopile was placed in the right antrum. In fifteen minutes the temperature had stabilized at 93.15° F. The lamp was applied in the usual manner. After one-half hour the lamp was turned off and the temperature had dropped 1.03° F. The readings were continued with the lamp off. The temperature continued to drop and in eight more minutes it had fallen an additional .96° F., making a total fall of 1.99° F. It then began to rise slowly.

It was necessary to remove the thermopile, at this time, on account of the fatigue of the patient.

#### *Experiment 9—Radiant Heat—Patient B*

The thermopile was placed in the left antrum. After eighteen minutes, when the temperature had stabilized, the lamp was placed in position in the usual manner over the left antral region. After thirty-five minutes the temperature had dropped 2.53° F.

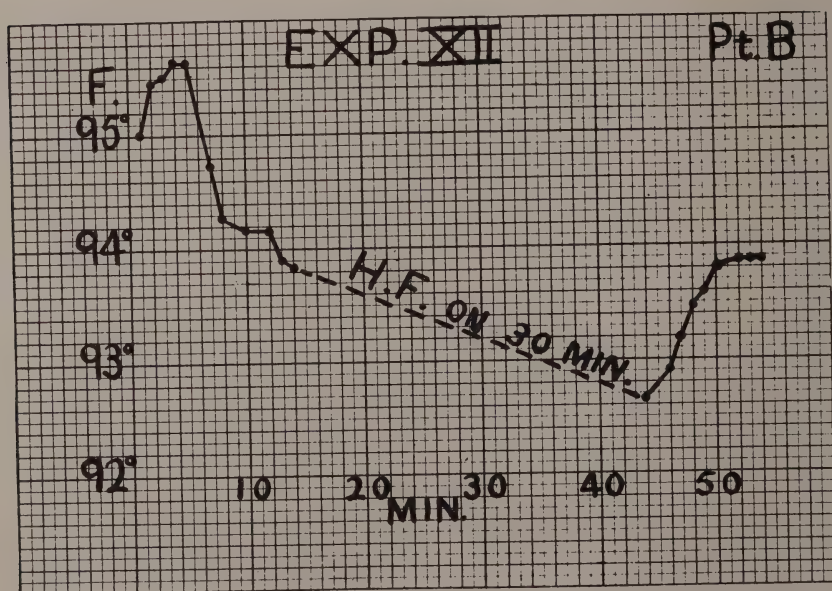
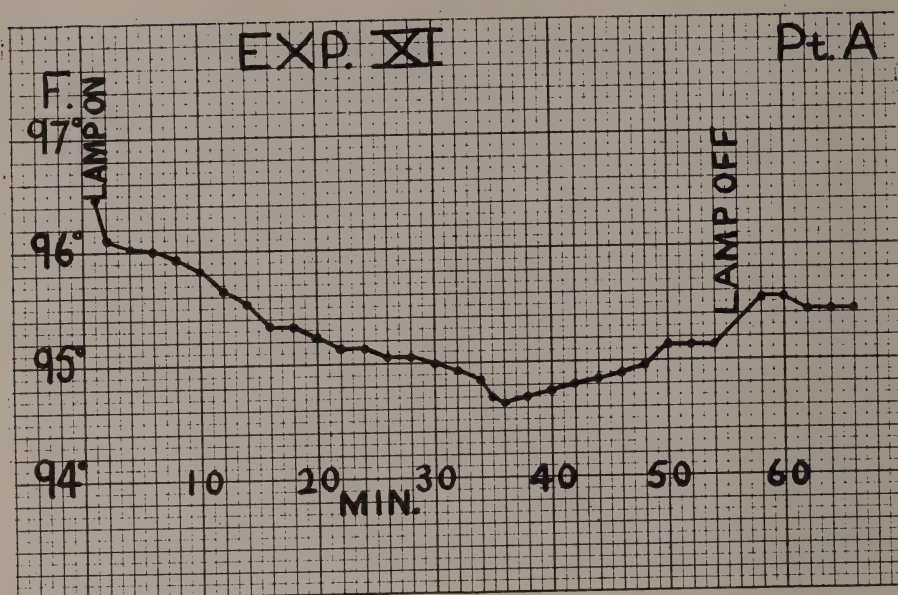
#### *Experiment 10—Radiant Heat—Patient A*

The thermopile was placed in the left antrum. After twenty-one minutes, when the temperature readings stabilized, the lamp was placed in the usual position. After the lamp had been on for twenty minutes, the patient had a severe paroxysm of coughing and the thermopile had to be removed. He then began to have a moderate amount of dyspnea, and the experiment could not be continued. In the twenty minutes, during which the lamp had been on, there was a drop of .5° F. The following morning, the patient developed a rather severe attack of asthma and was compelled to remain at home for eight days.

The patient's nose was examined before this experiment was begun and no sign of acute inflammation was noted. The mouth temperature before, during, and after the experiment was 98.4° F. The fact that there was only a limited drop in the temperature during this test might be accounted for by the fact that the patient was obviously on the verge of an acute upper respiratory infection.

#### *Experiment 11—Radiant Heat—Patient A*

The thermopile was placed in the left sphenoid. In fifteen minutes the temperature readings were well stabilized at 96.48° F. The lamp was put in place over the left side of the face. The temperature began to drop slowly for thirty-six minutes, when it read 94.65° F., a drop of 1.83° F. It then began to rise slowly, and after another thirty minutes it had risen to 95.42° F., a rise of .77° F. It was still 1.06° F. below the original reading.

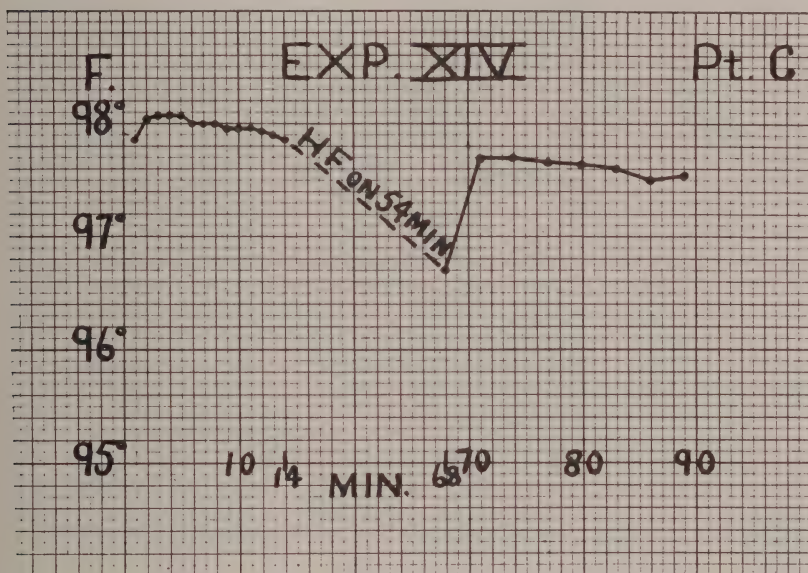


This drop occasioned considerable surprise as it was entirely unexpected. The thermopile was immediately recalibrated and the readings were found to be correct.

A moderate amount of muco-serous discharge was expelled from the nose.

*Experiment 12—High Frequency Oscillator—Patient B*

The thermopile was placed in the right antrum. It was allowed to remain there for fourteen minutes, until the temperature became stabilized at 93.85° F. The thermopile was then removed and the oscillator was turned on. After being on for thirty minutes it was turned off and the thermopile was quickly replaced in the right antrum.



When the readings became constant, the temperature registered 92.66° F. It remained there for several minutes, and then began to rise. At the end of ten minutes, the temperature had returned to the original reading of 93.85° F. This was a drop of 1.19° F. after thirty minutes exposure to the high frequency field.

The general reaction was very mild in this experiment. No sweating was noted, and only a moderate amount of nasal discharge as compared to the profuse secretion caused by the radiant heat.

*Experiment 13—High Frequency Oscillator—Patient A*

The thermopile was inserted in the left antrum. In twelve minutes the temperature registered 93.14° F. and remained stable at that point. The thermopile was removed and the oscillator was turned on for thirty minutes. The thermopile was replaced in the left antrum. The galvanometer went off the scale to the right, or red side, and would not return. It was left in the antrum for fifteen minutes but it would not register. It was removed from the

antrum and recalibrated. There was a drop of five and one-half full divisions in the calibrations. Search was made for the trouble and a short circuit was found in the thermopile. The result of this experiment could not be determined. A reserve thermopile was used in the subsequent experiments after many calibrations.

*Experiment 14—High Frequency Oscillator—Patient C*

The thermopile was placed in the right antrum. After fourteen minutes the temperature stabilized at 97.86° F. The thermopile was withdrawn and the oscillator turned on. After running for fifty-four minutes, the current was shut off and the thermopile was quickly reinserted in the right antrum. The temperature registered 96.7° F., a drop of 1.16° F.

*Experiment 15—High Frequency Oscillator—Patient A*

The thermopile was placed in the left sphenoid. After ten minutes the temperature had stabilized. The thermopile was removed and the oscillator was turned on. After thirty minutes heating, the current was turned off and the thermopile was replaced in the left sphenoid. No change in the temperature was noted. The patient felt no sense of general warmth, and the nasal secretion was very slightly increased.

## DISCUSSION

It appears, from the experiments described, that cold, derived from ice compresses and the ice bag, and that heat, derived from hot compresses, the hot water bottle and the electric pad, have essentially no effect upon the temperature of the sinuses when they are applied over the face. These physical agents do not appear to materially increase the nasal secretions nor to increase the moisture on the skin by sweating. On the other hand, the heat derived from the high frequency oscillating current and from the heat lamp used in these experiments apparently does cause a drop in the temperature within the sinuses and does produce an increase in the production of sweat on the skin surfaces. This is particularly true of the lamp. None of the agents used caused a rise in the body temperature.

It was not a surprise to find that certain of these agents had no definite reaction when applied, but it certainly was a great surprise to see the temperatures fall when the other agents were used. It was anticipated that the lamp and the high frequency current would, undoubtedly, increase the temperature within the sinuses, for there is ample experimental evidence to prove that heat from these agents does penetrate solid structures. Why, then, does it cause exactly the reverse to happen in the sinuses? It has been pointed out in this paper that an increase in the nasal secretions accompanied the use of the lamp and the oscillator. These two agents are the same ones



which cause a drop in the temperatures. Does it not seem reasonable to assume that the heat loss in these instances was due to the evaporation of an increased quantity of moisture? When the body is heated above a normal temperature, the skin surfaces are moistened by sweat. Is it not a well known fact that the respiratory tract also aids in the regulation of body temperature by dissipation of heat from its moist surfaces? This is particularly true of the dog and other animals who depend almost entirely upon this mechanism for reducing temperatures above normal. When all these facts are considered, it does not seem quite so strange that the temperature within the sinuses should drop when certain forms of heat are applied over the face. It appears, then, that this phenomenon is apparently a part of the mechanism involved normally in the regulation of body temperature.

It is not contended that the heat from the lamp and the high frequency oscillator actually reaches the membranes lining the sinuses, although there is a possibility that it might, for the same phenomenon might easily occur if the soft tissues, external to the sinuses, were heated and a portion of the excessive heat was dissipated from the body by way of the respiratory tract.

What about the practical application of these findings? Does it necessarily follow that because the temperatures dropped when the lamp and the oscillator were used that they have no value in the treatment of sinus disease? It is the opinion of the writer that these two agents are of great value in acute and subacute infections of the sinuses for it has been shown that there is a very definite increase of thin serous discharge from the nose and sinuses when they are used. This obviously improves the drainage, and hence hastens resolution.

#### SUMMARY

1. Cold compresses, ice bag, hot compresses, hot water bottle, electric heating pad, and an infra red heating lamp were applied over the sinuses, and the whole head was heated in the field of a high frequency oscillating current of 9,000 to 12,000 kilocycles.

2. Readings of the temperature changes within the maxillary and sphenoid sinuses were made.

3. No changes in the temperatures were noted when ice compresses, ice bag, hot compresses, hot water bottle, and the electric heating pad were used.

4. A drop of about a degree in the temperature of these sinuses occurred when the infra red lamp, and the high frequency oscillator were used. This drop was unexpected, but was accounted for by the

evaporation of the increased nasal secretions which accompanied the use of these agents.

5. The relative therapeutic value of these agents in the treatment of sinus disease is discussed.

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MEETING OF THE EASTERN SECTION HELD IN PORT-  
LAND, MAINE, JANUARY 4, 1935, UNDER THE CHAIR-  
MANSHIP OF DR. FREDERICK T. HILL OF WATER-  
VILLE, MAINE.

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POSTOPERATIVE BILATERAL ABDUCTOR PARALYSIS

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A great deal has been written, from both a clinical and an experimental point of view, on paralysis of the vocal cords. Paralysis of one cord is a rather common occurrence, usually resulting either from some pathologic lesion or an operative procedure. Indeed it has been noted that about 1 percent of persons examined in routine preoperative procedure for conditions not related to the thyroid gland have unilateral paralysis of the vocal cord.<sup>8</sup> The causes of this distressing, but not dangerous, condition are legion.

In bilateral abductor paralysis there is a loss of function of both crico-arytenoideus posticus muscles which normally abduct the cords, open the glottis, and permit free passage of air into the lungs. It is a comparatively rare disease, more common in men than in women and almost foreign to children.<sup>6</sup> It may be either central or peripheral in origin. If a lesion is central in origin, only partial paralysis will result. Peripheral paralysis is probably always complete in the case of a single laryngeal nerve just as it is with other nerves. The resultant laryngoscopic picture is easily explained by unopposed action of muscles supplied by other laryngeal nerves. True peripheral abductor paralysis occurs as the result of a lesion minute enough to involve only abductor fibers at their distribution in the larynx. The fact that there are four separate nerves supplying the larynx probably accounts for the confusion concerning paralytic lesions there.<sup>7</sup> Crile once said unilateral abductor paralysis is unfortunate, but bilateral abductor paralysis is a tragedy.<sup>2</sup> It is with this disease, as it follows operative procedure, that my paper deals.

The necessity of laryngeal examination before and after an operation, whether it be a thyroidectomy or some other operation on the neck in the region of the recurrent laryngeal nerves, cannot be too strongly urged. Not only is it a comfort to the surgeon to know

the condition of the larynx in advance of and subsequent to the operation, but such knowledge also has a beneficial effect on the patient. Should paralysis of one cord be detected before the operation, the operative procedure may possibly be altered to prevent bilateral abductor paralysis. The ancient adage concerning the ounce of prevention and the pound of cure is more than applicable to this condition, as prevention is, of course, the ideal treatment.

The most common cause of this form of paralysis following operation is, I feel, the pull on the recurrent laryngeal nerve during the process of removing the thyroid gland. Injury from forceps, knife or sponging is not so frequent, though these possibilities should be kept in mind. Any direct trauma during operative procedure, or direct pressure by, or involvement in, a pathologic condition in the region where the nerves are located, is likely to produce this condition. The capsule and the nerve are in close proximity and the latter can easily be traumatized. This fact is borne out by the report of Fowler and Hanson,<sup>3</sup> who found in their examination of 200 cadavers that the posterior capsule of the thyroid gland left the gland at the posterolateral portion of its lobe and passed directly backwards to the prevertebral fascia, thus leaving the posterior surface of the thyroid lobe uncovered and in direct contact with the nerve. It is the posterior margin of the thyroid, lying between the capsule and the nerve, that must be preserved and protected during the operation.<sup>2</sup> It must be remembered that this nerve, which is a branch of the vagus and supplies all of the intrinsic muscles of the larynx except the cricothyroid muscle, is very sensitive and delicate, one of the most vulnerable in the body, and is incapable of withstanding trauma of any kind, as some of the other nerves can. Pressure or pull interferes with nerve conduction and paralysis quickly results. This may be of two kinds: partial and temporary, or complete and permanent. During the past seven years I have seen eight cases of bilateral abductor paralysis resulting from operative measures, seven following thyroidectomy and one after removal of a gland complicated by extensive infection and scar formation. Of these cases four were temporary and four showed no improvement after eighteen months or more. It is the interest and desire of the surgeon to assure himself and his patient that he has placed his case in the former class by means of measures taken to afford immediate relief. Paralysis resulting from scar formation usually appears late and may be relieved if it is not allowed to continue for too long a period of time. If the condition has existed for more than twelve months there is little chance of recovery. The crico-arytenoideus posticus muscle, after loss of function for a cer-



tain time, will atrophy and will not return to normal function. This is one reason why prompt relief is desirable.

It is the distressing symptoms produced by bilateral abductor paralysis which make it essential for the surgeon to devise a speedy measure of relief. I recall two cases which I saw a few years ago and diagnosed as bilateral abductor paralysis following thyroidectomy. The surgeon in each case was not satisfied with my opinion and declared the condition due to "hysteria." Alarming complications developed but little attention was paid to them. One of those cases proved to be temporary and the patient recovered. The other was less fortunate and died four days later, very suddenly, before aid could be administered.

What, then, are the indications pointing to the condition? We find definite hoarseness and some stridor. Later, hoarseness is less noticeable and stridor more marked. Especially is this noticeable with increased excitement or with exertion. A peculiar noise presents itself on inspiration, especially during sleep. Expiration is quite natural and unchanged. The patient dreads the attacks, and in time, unless relief is secured, becomes more or less of an invalid. Dyspnea increases as the voice, which may in time return almost to normal, improves. Direct laryngeal examination during cordal paralysis reveals cords and arytenoids in midline with only a narrow space between the cords. Their position is straight because the tensor function is not resisted. On inspiration the cords are drawn downward and inward, preventing the intake of air. On expiration the cords are forced upward by the outgoing current of air and there is a separation of the cord, more marked in the middle. There have been some cases reported in which the cords are in a cadaveric position which does not interfere with breathing but where the patient would still have difficulty in speaking. Personally I have not, in my limited experience, encountered any such cases.

The treatment begins with immediate tracheotomy, which alleviates the danger of asphyxia, removing the strain from the heart and affording time for study of the type of surgical procedure to be undertaken. If the case proves to be a temporary paralysis and the function of the cords returns, the tube can be removed and the opening closed without any ill effects. On the other hand, if sufficient time elapses to ensure that the condition is complete, one of the several types of operations should be selected and performed.

There are various kinds of surgical procedure, some successful and some which have failed in nearly every case. It was early found that cordectomy offered little or no relief. Jackson improved on this

when he introduced ventriculocordectomy. This operation, while quite successful produced a grave impairment of voice, which was, of course, less serious than the imminent danger of asphyxia. The operation devised by MacKenty<sup>6</sup> is not a difficult one, and it gives the necessary relief and at the same time preserves the voice. This operation aims to establish a small permanent opening in the trachea at a point just above where the trachea dips backward into the chest—the success of the procedure depends on securing a union between the skin and the mucous membrane, entirely free from scar tissue—the air passing through this small opening in the trachea adds to the air inspired through the natural breathway and counteracts the inspiratory pull on the cords. This pull is the chief factor in reducing the amount of inspired air. The stronger the pull the less air inspired. The opening also acts as a safety valve during the dangerous spasms of asphyxia so common in abductor paralysis. Should it be found, after this operation has been performed, that the normal function of the cords has returned, it is a simple matter to restore the larynx to its preoperative condition. This procedure may well be used as an intermediate step between tracheotomy and ventriculocordectomy which may be performed later. Indeed, where time and the condition of the patient permit, it may well supplant tracheotomy entirely.

Nerve anastomosis has been tried but with little success. Another type of operation is submucous resection of the cord. This has been tried by many with varying success. Hoover,<sup>4, 5</sup> of the Lahey Clinic, reported several operations of submucous resection which have been quite satisfactory. An ample airway is established and though normal voice is impaired a good whispering voice is assured. The object of this procedure is to widen the lumen of the larynx by the removal of soft tissue between the mucous membrane and the cartilage of the lateral wall, thus allowing the mucous membrane to be placed on the lateral wall, increasing the lumen and preventing the formation of granulations. A tracheotomy is the first step in this procedure, if one has not already been performed. An important point to be emphasized is that all tracheotomies should be done below the cartilages entering into the fundamental structure of the larynx, preferably below the second ring of the trachea. To make the opening higher than this is to invite a possible permanent dependence on a tube.

In summary, let me stress once more the importance of a laryngoscopic examination before and after thyroidectomy. Should the paralysis present itself following operation, treatment is indicated by the condition of the patient.

## REPORT OF CASES

*Case 1.*—Mrs. D. I., aged nineteen, a housewife, was seen in consultation, complaining of nervousness, tremor and attacks of asphyxia. Six months previous to this a thyroidectomy was performed. Convalescence was slow. For two months she had a whispering voice, which improved but nervousness and asphyxia became more marked until the attacks were alarming. She was put on calcium therapy, advised to rest, and tracheotomy was performed. Indirect laryngeal examination showed both cords and arytenoids motionless and in midline. Her condition improved, except for difficulty in breathing without means of a tube. Cords have remained in midline with no evidence of recovery of paralysis after eighteen months.

*Case 2.*—Miss C., a school teacher, following an operation for removal of glands and extensive infection, later developed dyspnea. Attacks of dyspnea became alarming and she was admitted to the hospital for observation. Indirect examination revealed a bilateral paralysis. There was no improvement after several months and under intratracheal anesthesia scar tissue was resected from region of recurrent nerve. Following this operation there was a gradual return of cords to normal movements.

*Case 3.*—Mrs. D., aged thirty-six, presented herself to bronchoscopic clinic for shortness of breath. Three months previous she was operated upon for thyroidectomy, from which, as she stated, she never completely recovered. At first she had hoarseness which cleared up, but had attacks of dyspnea and nervousness. Indirect examination showed paralysis of both cords. Following tracheotomy she was relieved of the distressing symptoms. After one month the cords were in midline. Further observation was not possible. This case had not been examined before or after operation until she came to the clinic.

*Case 4.*—Miss D., aged twenty-six, was operated upon for thyroidectomy. Immediately following there was hoarseness with some dyspnea. Laryngeal examination showed complete paralysis of the left cord. A few days later there was paralysis of the right cord, with stridor. The voice improved and likewise the condition. After a few weeks paralysis had disappeared.

*Case 5.*—Mrs. B., a housewife, was being operated upon by Dr. C. W. Maxson for substernal thyroid. Suddenly she became cyanosed and was unable to inspire. Immediate direct examination revealed both cords in midline. A hard rubber French catheter was inserted between the cords into the trachea. Her condition immediately improved and they were able to continue with the operation. The catheter was removed late the same day. There was some dyspnea for a few days, but paralysis gradually disappeared and she made a good recovery. Comment: This condition was due to bilateral paralysis, due to pulling on nerve in delivering gland. The insertion of catheter permitted operation to be finished and possibly saved the patient's life.

*Case 6.*—Mrs. K., aged forty-one, housewife, was seen in consultation on account of stridor following thyroidectomy three months previous. Following removal of the thyroid she was hoarse for six weeks. When this improved the stridor increased in severity. Indirect examination showed both cords in midline. There was a very narrow space in the glottis between cords, with no abductor motion. The obstruction during the attacks was so severe that there was retraction of the sternal notch and intercostal space. There was no interference with expiration and the voice was fairly good. Immediate tracheotomy

was advised, but was not permitted by the surgeon, as he felt this was largely a nervous condition. Four days later she died suddenly before aid could be administered. Comment: Tracheotomy would have saved the patient's life. Hysteria does not produce objective or subjective symptoms such as this patient presented.

*Case 7.*—Mrs. M. R., aged fifty-six, a housewife, was referred to a clinic for laryngeal examination. She had had a thyroidectomy three years previous, following which she had nervousness and difficulty in breathing, especially on exertion or excitement. Examination showed she was wearing tracheotomy tube. She appeared well nourished. Basal metabolism was within normal limits. There was hypertension and cardiovascular disease of long standing. The patient was seen by Dr. Chevalier Jackson, who made the following suggestions: that she wear a valve canula which would allow comfortable breathing and talking; that a partial cork used during the daytime be removed at night for comfortable breathing; that the outer canula be removed daily and cleaned and that the inner canula be removed and cleaned as often as necessary; that care be taken to see that the canula fitted perfectly, so as not to hinder free passage of air. Both cords were in midline and there was only a narrow space present between the ends on expiration. This was due only to the force of expired air. Because of the age of the patient and the duration of the condition she was advised to use a tracheotomy tube, since she was comfortable and able to talk without difficulty.

*Case 8.*—Mrs. W., aged forty-two, was admitted to Mercy Hospital, November 21, 1928, for enlarged colloid thyroid. Laryngeal examination showed both cords normal. Following thyroidectomy, under gas anesthesia, there was a short period of hoarseness and rapid pulse. This improved and dyspnea became more marked. Laryngeal examination revealed both cords in midline and no abductor movement. The patient was seen again on January 6, 1929, at which time the paralysis had completely cleared up.

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## THE MANAGEMENT OF MALIGNANCIES OF THE SINUSES

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The history of cancer of the sinuses is familiar. It was extant before the oldest medical records, and early treatment was futile. That courageous English surgeon who some three hundred years ago treated his king with an actual cautery for what was probably a cancer of the antrum undoubtedly carried out one of the first scientific efforts to combat this disease. Other sporadic efforts are recorded, but the scientific treatment of cancer is recent. With the development of modern surgical methods, extirpation became possible and was used with and without cauterization. As a life-saving measure, this was frequently adequate, but the problem of disfigurement and rehabilitation necessitated the development of plastic flaps and concealed incisions to render the cure less obnoxious. Moure, Denker and Grosgean entered the picture at this point. Later, Thomson, Tilley and others adopted, revised and modified the earlier methods and are still carrying on to the present day. The development of electrosurgery by Clark in 1907, and the discovery and application of radium and x-ray, however, have given the surgeon of today additional means of controlling this disease.

Cautery, surgical extirpation, endothermy, x-ray and radium represent the chronologic sequence in the treatment of malignancies of the sinuses, and they were employed in any combinations that were possible at one particular time. At present all are possible, and such men as Patterson, New, Beck, Guttman, Cade, Quick, Barnes, Ohngren, Greene and others offer us the fruits of their experience in testing these various combinations. Barnes and Ohngren feel that surgical extirpation followed by radium and x-ray, with subsequent plastic operations, if necessary, have been satisfactory. Beck and Guttman do not seem enthusiastic about surgical extirpation *per se*. Patterson has obtained excellent results with endothermy and supplementary radiation. I have no information concerning the results of New's 295 cases of malignant tumors of the upper jaw and antrum, but I believe he used diathermy and radiation. Before this, however, he has said: "The combination of cautery and radiation . . . has produced a greater percentage of cures than previous operative meas-

ures." Douglas Quick states that from a life-saving standpoint conservative surgery for access, drainage and radiation actually gives better results than can possibly be obtained by the most extensive surgical extirpation.

No laryngologist can extricate himself from the management of malignancies. If he refuses to handle them he will probably fail in their recognition and thereby commit an error of omission.

In determining the competency of the laryngologist to handle these cases, let us consider that he is a man of average surgical ability, that his first consideration is the welfare of the patient, that he desires to manage the case in accordance with the best medical and surgical practice, that he is willing to eliminate himself at any time if by so doing the interest of the patient is better served, and, further, that he wishes to conduct his operative diagnostic procedure so that further treatment follows as a natural sequence. It should be understood, however, that he is not a radiologist.

With these prerequisites we shall investigate nine cases of malignancies of the sinuses to which the author has had to apply himself within the last three and one-half years. We shall note the methods of attack, the manner in which the cases were managed, the results obtained and the problems raised. Included in this group are sinuses both primarily and secondarily involved. Briefly, the routine in handling the cases was as follows:

(A) *Typical:*

1. X-ray (preferably with lipiodol).
2. External opening into the sinus for biopsy, window, drainage and radiation.
3. Pathologic consultation concerning the type of growth and probable prognosis.
4. Radiation (with or without further diathermy as the case required).

(B) *Atypical:*

The routine in the atypical case was extirpation, surgical or diathermy, followed by radiation to meet the demands of that particular case.

With one exception, the operative procedures were done by the author in accordance with the requirements for postoperative radiation. The radium and x-ray treatments were given by local radiologists, the State Institute for the Study of Malignant Disease in Buffalo, Douglas Quick, Johns Hopkins Hospital and the Rochester General Hospital.

The pathologic studies were important and interesting. In every case tissue was sent to at least two recognized pathologists, and in one instance pathologists in six different medical centers examined the same tissue. A general agreement as to the degree of malignancy was evident, but borderline cases brought in varied reports and different opinions concerning treatment and prognosis. Case No. 8 illustrates this point.

CHART I.—CASE 8

<i>Pathologist</i>	<i>Diagnosis</i>	<i>Suggested Treatment</i>	<i>Prognosis</i>
J. Ewing, New York	Glandular type of growth, radiosensitive	Radiation	Good
C. V. Weller, Ann Arbor	Round cell carcinoma	Surgical extir- pation	Guarded
I. Gaspar, Rochester, N. Y.	Angiosarcoma	Radiation	Guarded
G. H. Whipple, Rochester, N. Y.	Highly malignant tumor, radiosensitive	Radiation	Fair
B. T. Simpson, Buffalo	Epithelioma	Radiation	Good

The results indicate that the assumption of Dr. Ewing and others was correct. Broders' method of grading cancer appeals to me as a surgeon, but I could get neither Ewing nor Weller to commit himself to any fixed formula for treatment or prognostication. The most important responsibility of the pathologist appears to be in the recognition of radiosensitive types.

Chart II gives a summary of the facts concerning these cases.

The present status of the patient in these nine cases after three and a half years is as follows:

Five are dead. One is not expected to live. In Case No. 7 (the giant cell sarcoma) the tumor is slow growing and continues to progress. Two patients (Cases Nos. 8 and 9) are presented as cured. Incidental information:

In two cases, or 22 percent, the diagnosis was unsuspected.

In six cases the disease was far advanced at the time of the first examination.

One patient died as a direct result of the operation.

Four died as a direct result of the malignancy.

CHART II

<i>Case</i>	<i>Diagnosis</i>	<i>Involvement</i>	<i>Treatment</i>	<i>Present Status</i>	<i>Longevity</i>	<i>Impression</i>	<i>Complaints</i>
1. M. B., Age 56, F.	Squamous cell carcinoma	Antrum, ethmoid, orbit	External antrum op., x-ray	Dead	9 months	Nasal discharge 6 years	Chronic maxillary sinusitis
2. W. G., Age 59, M.	Squamous cell carcinoma	Cheek, alveolar process and antrum floor	Complete extirpation, diathermy, x-ray and radium	Dead	3 years and 5 months 10 months after operation	Sore spot in mouth	Malignancy
3. W. F., Age 63, M.	*Macroscopic carcinoma	Nasopharynx, antrum, ethmoid, orbit	X-ray (inoperable)	Dead	1 year and 2 months	Nose bleeding, deafness, pain	Anemia, malignancy
4. L. B., Age 65, M.	Round cell sarcoma	Frontal orbit and general metastasis	External frontal operation	Dead	7 months	Pain in back, bulging eye	Mucocoele, question of malignancy
5. W. P., Age 73, M.	Infiltrating squamous cell carcinoma	Antrum, cheek, orbit, malar region	Complete extirpation, diathermy	Dead	1 day	Fistula in cheek	Malignancy
6. A. E., Age 53, F.	Trans. cell carcinoma	Antrum	External antrum op., x-ray and radium	Alive	1 year plus	Pain	Malignancy
7. B. V., Age 29, F.	Giant cell sarcoma	Jaw, antrum floor, mandible	X-ray	Alive	8 years	Loosening of teeth	Malignancy
8. H. G., Age 42, F.	Radiosensitive epithelioma	Antrum, and orbit	External antrum op., x-ray and radium	Alive	2 years and 6 months plus	Pain	Malignancy
9. M. G., Age 24, F.	Osteoma	Antrum	External antrum operation	Alive	3 years and 4 months plus	Pain	Chronic maxillary sinusitis

\* Specimen unsatisfactory.



One highly malignant tumor is presented as cured (Case No. 8, radiosensitive).

Two tumors (the osteoma and the giant cell sarcoma) are obviously not malignant in the same sense as the others.

Percentage rates of success or failure may be so varied as to be almost valueless. After three and a half years the percentage of successful results was 45 percent. If, however, we eliminate the osteoma and giant cell sarcoma, it is 16 percent.

### CONCLUSIONS

1. Too many of these patients died. If it had been permitted, should more radical extirpation have been done?

2. Primary neoplasms of the sinuses are usually seen late because the symptoms appear late.

3. Early operation for biopsy, access and drainage is indicated for diagnosis and may be sufficient to render further surgery unnecessary.

4. Pathologic consultation is necessary for the proper conduct of the case.

5. Subsequent surgery or diathermy should be employed only after special study.

6. Radiation should be relied upon as much as possible.

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## THE PRESENT STATUS OF SINUS INFECTION AS AN ETIOLOGIC FACTOR IN RETROBULBAR NEURITIS

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This old subject, concerning which volumes have been written in recent years, is still without any conclusive evidence as to the procedure to follow when confronted with such a problem.

It is with fear and trepidation that I approach the subject, but several articles have recently appeared in medical literature which gave me grave thought that the pendulum might swing too far from the sinuses as a causative factor in this condition, *i.e.*, retrobulbar neuritis.

Quoting from the late Ross Hall Skillern, "The intimate relation of the sphenoid sinus with the optic nerve is a predisposing factor to retrobulbar neuritis following sphenoid sinusitis, particularly if a dehiscence is present. No regularity exists as to the thickness of bone separating the sinus from the nerve. Investigations have shown that these measurements undergo great variations in different heads and even on different sides of the same head, as in one instance the nerve may be almost in direct apposition to the mucosa of the sinus, while on the opposite side several mm. of spongy bone may intervene."

The posterior ethmoid or sphenoid cells are also in very close proximity to the nerve.

A number of observers have examined a series of patients with sphenoiditis for signs of involvement of the optic nerve without any complaint on the part of the patient of ocular symptoms. Markbreiter found that 70 percent of a series showed defects in the fields of vision, and Bordley found enlarged blind spots in 31 of 102 patients examined.

With this evidence before us, it is reasonable to assume that we are not dealing with a possibility as remote as some authors would have us believe.

Von Hippel<sup>1</sup> asks the question, "Are the successes of nasal therapy actually the result of nasal operations or are they produced by spontaneous recovery?" He believes the latter to be the case. He bases his conclusions on a series of thirty-six cases; in thirty there was no pathologic condition in the sinuses by roentgen ray examination.

The remaining six cases were definitely either multiple sclerosis or hereditary optic atrophy. Twenty-one cases recovered; the remainder pursued an unfavorable course or failed to report. None were operated upon; thus surgery was prevented from scoring a 70 percent success.

Benedict<sup>2</sup> states: "When one considers the vast number of cases of severe suppurative sinus disease without visual symptoms the connection between sinus disease and retrobulbar neuritis becomes much less credible."

He gives in support of his opinion an analysis of the etiology of 225 cases of retrobulbar neuritis at the Mayo Clinic, in which series but one case was considered as due to sinus disease. He believes that the therapeutic value of operation on the sinuses does not lie so much in the eradication of the infected area as to the immediate ischemia from blood letting followed by a secondary congestion of the adjacent tissues until healing is complete. "If the operation has been sufficiently extensive, there is commonly a rise of temperature of one to two degrees from the absorption of blood which, in effect, is auto-vaccination. These two factors, congestion and absorption, are responsible for any improvement noted."

Blatt<sup>4</sup> found the number of cures obtained in patients on which operation was performed was no greater than the number obtained in patients on which no operation was performed. Nor was the course of the disease influenced by operation. He advises surgical intervention in apparently negative cases where no results have been obtained by other means after a period of two to three weeks.

However, let us contrast a report by Bajkay,<sup>3</sup> who reviewed forty cases in which the ethmoid and sphenoid sinus were opened, where rhinologic examination gave positive results in twenty-two instances and negative in eighteen. In both of these groups operation produced a complete cure in those patients who entered the clinic in from three to four days after the onset of visual disturbances, but in those who entered between the eighth and fourteenth day after onset of symptoms only a partial restoration of vision could be obtained. In eight cases there had been a delay of from three to four weeks before admission and no improvement resulted.

Bachstetz<sup>5</sup> states that while rhinogenous retrobulbar neuritis is not rare, rhinogenous cases with negative rhinologic findings are rare.

There have been cases immediately following epidemics of influenza in which multiple sclerosis could be definitely ruled out. Feigenbaum and Salzburger<sup>6</sup> reported a series of twenty-four cases, twelve of which showed x-ray evidence of sphenoid and ethmoid sinus infec-



tion. Sixteen were operated upon; of these, eleven were cured. The fact that the major portion of these cases occurred about four weeks following influenza seems to be reason for considering that the etiology was nasal in this particular group.

In order that I might have the most recent opinion of leaders in ophthalmology and rhinology in this country, I addressed an inquiry to men in various sections. As might be expected, those agreeing that sinusitis was an important factor in retrobulbar neuritis were, for the most part, rhinologists and to a lesser number ophthalmologists. Each man was asked for his present attitude in regard to the etiologic factor of sphenoid infection in retrobulbar neuritis.

O'Brien of Iowa City said: "I have seen very few cases of retrobulbar neuritis which I feel were due to sinus disease. I am not satisfied that an operation on the sinuses is indicated in those cases which have slight evidence of disease."

Edward Jackson of Denver answered: "In cases of monocular neuritis I have not advised opening of the sinuses; and in no case has the second eye been subsequently involved."

Weymann of Los Angeles replied: "Today I would feel that permanent damage to the optic nerve is not accomplished so quickly as one might think, so that in the absence of evidence of disease of the nervous system, specific disease, or other frank focal infections, I should still be inclined to wait a couple of weeks before opening the sinuses if there was no great evidence of involvement; and then only with the idea of producing a reaction in the region of the optic nerve which might lead to its recovery, rather than by the elimination of infection present in the sinus."

Lancaster of Boston reported no recent cures by operation on the sinuses but agreed that where all other neurologic evidence is negative an operation on the sinuses might benefit because of the hyperemia which it produces.

Friedenwald of Baltimore replied in part as follows: "I do not feel that in the great bulk of cases the relation of sinus disease to retrobulbar neuritis has been established. However, I have seen two cases in which I felt sure the sinus infection was definitely related to the ocular symptoms. Both of these patients had slight exophthalmos, pain on pressure over the globe, pain on ocular movement, and a purulent infection of the ethmoid and sphenoid sinuses on the side of the ocular trouble, and both recovered promptly after drainage of the sinuses."

There were, however, two reports from leading ophthalmologists that tended to support the ideas of the responding rhinologists.

Berens of New York said: "I have found a number of cases improved or cured by operation, but I do not advise surgery until local nasal treatment and the use of specific and non-specific antigens for varying periods have been unsuccessful."

Peter of Philadelphia was very enthusiastic in his support of operation and I quote his reply at length: "I find a very large number of cases of retrobulbar neuritis due to sinus disease. In some instances the cure can be established by medical means, but in the majority of instances the retrobulbar neuritis is converted into frank optic neuritis and papillitis, and in some instances a papilledema, if operative interference is not practiced.

"Such papers as were presented from the Mayo Clinic are very misleading. Either these men see none of the acute cases or their observations have not been very accurate. I have thought it was the former reason.

"The subject is very timely and a most important one to bring before the profession, because it will give an opportunity of stressing the sinuses as the most outstanding factor."

The reports from rhinologists were more in accord.

Beck of Chicago answered as follows: "Merely from memory I recall seventeen cases of marked loss of vision, usually more in one eye than in the other, in which ophthalmologic as well as the rhinologic findings were practically negative. However, the blind spot was enlarged in most of them. I have observed three of these cases long enough now to state positively that they were progressive multiple sclerosis. Two others were alcohol-tobacco amblyopia."

Fenton of Portland, Ore., replied that he could recall offhand several cases which had been cured by operation. He feels that where rapid loss of vision is taking place the patient should receive the benefit of the doubt and decongestive measures involved in improved posterior sinus drainage should be made use of.

Hurd of New York has had very brilliant results but there was evidence of sinus trouble with good x-ray films.

Ridpath of Philadelphia: "In spite of the diversity of opinion between the ophthalmologists and the sinuologists, it is my opinion that infections of the postethmoid and sphenoid cells can be, and are, a decided factor in retrobulbar neuritis."

In 1932 I reported before the New Jersey State Medical Society two cases of sinus origin, in both of which I obtained almost complete cure by operation.

How can we settle this question? In fact, how can any disputed question in medicine be settled? Apparently it will always be one of the medical riddles, and each rhinologist will be forced to use his best judgment in deciding what procedure to follow.

There does seem to be proof that the optic nerve can be affected to a mild degree in quite a percentage of cases. Then why not in the occasional case in which we are particularly interested?

With the present knowledge of sinus anatomy and operative dexterity that most well equipped rhinologists possess, operation on the sphenoid and ethmoid sinuses should not be such a dangerous procedure. An occasional needless operation on a case later proven to be multiple sclerosis cannot begin to hold for us the chagrin that we would feel over a blind patient whose sight might have been saved had we been bold enough to insist on operation at the time when sight might have been restored.

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# EXPERIENCES IN IONIZATION OF THE NASAL MUCOUS MEMBRANE

By HAROLD G. TOBEY, M.D.

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The theory of ionization of mucous membranes by metallic solutions aided by an electrical current has been applied for more than a decade, but has not been widely practiced. A new impetus has been given to this therapeutic measure through the work of H. L. Warwick, who has investigated the results to be obtained from various metallic solutions and their combinations, and has developed a means by which a definitely controlled smooth direct current of electricity may be passed through the structures to be treated without harm to the tissues or discomfort to the subject.

The theory, briefly stated, is that a metallic solution placed in contact with the nasal mucous membrane results in the precipitation of the proteins of the superficial cells, but when a mild direct current is passed through the tissues the metallic ions penetrate more deeply and precipitate more of the proteins of the cells. Great care is taken that the strength of the current does not reach the point of coagulation of the tissues.

Warwick, after considerable experimentation, has adopted a solution consisting of sulphate of zinc 85 percent, tin 10 percent and cadmium 5 percent, as best suited for the treatment. An alloy of these three metals, in the same proportions, is used for the anode.

The method of treatment consists in thoroughly anesthetizing the entire mucous membrane, following which, the nasal cavities are thoroughly packed with cotton strip saturated with the metallic solution and the anode introduced. The cathode is then affixed to the arm of the same side. The efficiency of the packing is indicated by a resistance ohmmeter which is incorporated in the instrument. The resistance should be from 2,500 to 3,000 for a satisfactory result. If this resistance should be as high as 8,000 to 10,000 ohms, it is positive evidence that the nasal packing is inadequate.

The direct current is then turned on slowly to the desired strength. One hundred (100) milliampere-minutes—that is, ten milliamperes for ten minutes—has been found to give good results. However, the strength of the current may be varied according to circumstances, and as dictated by experience.



The immediate discomfort to the subject is negligible and consists of having the nasal cavities tightly packed and a metallic taste as the current reaches its maximum. The after results are more troublesome. Soon after the completion of the treatment there ensues a period varying, usually, from eight to twelve hours, when there is an intense burning sensation in the nose, eyes and face accompanied by some pain and headache. In addition there is frequently a marked nervous reaction, the extent of which is dependent on the temperament of the subject.

The treatment has been developed primarily for the alleviation of hay fever and seems to be particularly applicable to those vasomotor disturbances of the nasal mucous membranes which are today usually classed as allergic.

Our experience, so far, has consisted of the treatment of sixteen subjects with a conservative general estimate of 85.6 percent of relief.

Eight of these suffered from hay fever and in this group there was 85.6 percent of relief. One subject suffering from fall hay fever was treated in June with the result that he was entirely free for the first half of the season, after which the usual symptoms intervened with their usual severity. All patients treated after the onset of the symptoms were free of all discomfort thereafter.

Eight patients were the victims of a hyperesthetic rhinitis and, among these, three were also sufferers from asthma. Two of the latter had already been relieved of asthma before the treatment was instituted, while in the third there was no relief following the treatment. In this group it will be noticed that while there was 83.7 percent of immediate relief, the time element begins to make itself manifest.

In Case 10 the patient had suffered from asthma, urticaria and hyperesthetic rhinitis for two years when first seen two years ago. She had been relieved of asthma now for one and a half years, and of the urticaria for one year. All efforts at relieving the rhinitis had proved of no avail up to May, 1934, when the Warwick treatment gave complete relief for six months. The second treatment has just been completed so that the result is not yet available. The causative agent seems to be heavy perfume, which she has no way of avoiding since her livelihood depends upon the teaching of music.

In Case 11 about 50 percent relief was experienced for a period of two months.

In Case 9, 50 percent relief for the full six months followed the treatment.

The other patients have experienced immediate relief, but a sufficient time has not as yet elapsed for any final decision.

The change in the appearance of the mucous membrane is as striking as the relief from symptoms. Following the stage of primary congestion, the mucous membrane takes on the color of normal healthy lining, which is in great contrast to the pale, boggy appearance before treatment.

Case	Age	Dates of ionization	Indication for ionization			Duration of symptoms	Result of ionization	Remarks
			H. F.	H. R.	Asth.			
1	23	June '34	*	..	..	2 yrs.	50%	Fall hay fever treated in June
2	60	Sept. '34	*	..	..	40 yrs.	100%	.....
3	40	Aug. '34	*	..	..	....	100%	.....
4	55	Aug. '34	*	..	..	....	85%	.....
5	7	Oct. '34	*	..	..	5 yrs.	75%	.....
6	12	Oct. '34	*	..	..	3 yrs.	100%	.....
7	55	Sept. '34	*	..	..	....	100%	.....
8	16	Aug. '34	*	..	..	10 yrs.	90%	.....
9	50	May '34	..	*	..	10 yrs.	50%	.....
10	48	May '34	..	*	*	4 yrs.	100%	Asthma, urticaria H. R. relieved 6 mos.
11	43	Aug. '34	..	*	..	3 yrs.	50%	Two months' relief
12	45	Oct. '34	..	*	*	17 yrs.	100%	No relief to asthma
13	20	Aug. '34	..	*	..	.3 yrs.	75%	.....
14	18	June '34	..	*	..	3 mos.	100%	.....
15	52	Aug. '34	..	*	*	10 yrs.	95%	Asthma, Migraine, Urticaria
16	40	Nov. '34	..	*	..	3 yrs.	100%	.....
			8	8	3			

### CONCLUSIONS

1. The method is efficient for the control of hay fever if instituted after the onset of the symptoms. We feel, however, that desensitization should be the method of choice.

2. The immediate results in hyperesthetic or vasomotor rhinitis are excellent. The duration of these results is as yet unknown.

# STUDY OF AUDITORY FUNCTION IN 100 PUPILS AT THE CLARKE SCHOOL FOR THE DEAF\*

By RUTH P. GUILDER AND LOUISE A. HOPKINS

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Detailed studies of auditory function and careful analyses of the resulting audiometric findings in pupils in schools for the deaf are becoming increasingly important. Such studies carried out over long periods of time and under carefully controlled conditions should add to the existing knowledge of deafness in childhood; coupled with studies of auditory function in other members of the family, they should form the basis of a program for the prevention of future deafness; and, most important of all from the standpoint of the child already so seriously handicapped, they should help materially in the more scientific development of the educational program for the deaf child.

Many of the studies which have been made in the past have failed to give a complete picture of the auditory function of such groups: (1) because of the impossibility of obtaining an accurate picture of the auditory function of a deaf child at a single test, if his sense of hearing has not been previously trained; (2) because of the inadequacy of the audiometers in use in most schools for the deaf to explore the entire auditory field; and (3) because many of the audiometric studies have been based on air conduction alone rather than on a comparison of the results obtained by both air and bone conduction under carefully controlled conditions.

It is hoped that some of these difficulties may be obviated in the audiometric studies which are now being made. At The Clarke School the average child may be studied over a period of ten years. A program has been devised for the training and testing of hearing in the preschool or entering child, and emphasis is placed on the use of the child's residual hearing throughout his school years. Hearing tests under physiological conditions are carried out regularly at six-month intervals or oftener. In a previous paper, a survey of the changes in the hearing curves over a period of three years has been recorded. All records used for the present study have been made on a modified and greatly amplified 2-A audiometer, which makes possible the exploration of practically the entire auditory

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\* From the Research Department of The Clarke School for the Deaf, Northampton, Mass.

field in the deaf child by both air and bone conduction. For these reasons, we hope that the audiometric studies which have already been under way at The Clarke School for the past four years may make some small contribution to the scientific knowledge of deafness in childhood, to the educational program for the deaf child, and eventually to the development of still more active prophylactic measures.

In the present paper, we wish to record our first detailed analysis of the general trend of the hearing curves by both air and bone conduction in a typical group of 100 pupils, the trend of the hearing curves for the main etiological subgroups, and a comparison of our findings by air and bone conduction both for the entire group and for the etiological subgroups. In a subsequent series of papers, a further analysis of air conduction, bone conduction, and vestibular findings for the same group will be recorded, the present paper being in the nature of a general survey of auditory function in such a typical group, with the conclusions which may be drawn therefrom.

#### DATA AND TECHNIC

The study consists of a statistical analysis of the audiometric readings made by air and bone conduction on 100 pupils at The Clarke School for the Deaf in the fall of 1934. Mean curves and frequency distributions at the various pitches for both air and bone conduction have been established for the entire group and for the etiological subgroups; mean curves for air and for bone conduction have been compared, and the mean differences between the air and bone conduction readings for the entire group and for the subgroups have been compared with the mean differences between air and bone conduction readings in a normal group.

The pupils included in this study were unselected as to cause of deafness or amount of residual hearing, and constitute the entire population of the upper and middle schools during the year 1934. The group is fairly representative, both as to cause of deafness and character of hearing loss, as compared with similar groups studied during the past four years and may, therefore, be considered a fair sample of Clarke School's older pupils. For obvious reasons, audiograms of pupils in the lower school are not included in scientific analyses, although increasingly accurate responses are being obtained from even the youngest group.

Etiologically, the group is constituted as follows: deafness of congenital origin, 50 percent; deafness of probably congenital origin, 16 percent; deafness due to cerebrospinal meningitis, 14 percent;



deafness due to primary disease of the ears, nose and throat, 12 percent; and deafness due to a toxic neuritis of the auditory nerve following an acute infectious disease, 8 percent (Table I). We are accustomed to classify as "probably congenital" deafness recognized prior to the acquisition of speech when both parents and physicians are in doubt as to whether it has been present since birth or has resulted from some slight illness during infancy.

TABLE I.—ETIOLOGICAL CLASSIFICATION OF 100 UNSELECTED PUPILS INCLUDED IN THIS STUDY

<i>Cause of deafness</i>	<i>Number of pupils</i>	<i>Percentage</i>
Congenital .....	50	50
Probably congenital .....	16	16
Cerebrospinal meningitis .....	14	14
Disease of ear, nose and throat.....	12	12
Toxic neuritis .....	8	8
Total .....	100	100

In age, the group ranges from nine to twenty years (Table II).

TABLE II.—AGE DISTRIBUTION ACCORDING TO ETIOLOGICAL CLASSIFICATION

<i>Cause of deafness</i>	<i>Age at nearest birthday</i>												<i>Total</i>
	9	10	11	12	13	14	15	16	17	18	19	20	
Congenital .....	1	1	3	5	7	9	9	6	3	2	3	1	50
Probably congenital ..	..	..	..	1	3	1	2	4	2	2	..	1	16
Cerebrospinal meningitis .....	..	..	1	3	..	1	..	2	3	2	..	2	14
Disease of ear, nose and throat .....	..	..	1	1	1	1	1	3	1	1	1	1	12
Toxic neuritis .....	..	..	1	1	1	1	2	1	..	1	..	..	8
Total .....	1	1	6	11	12	13	14	16	9	8	4	5	100

Boys constitute 46 percent of the group, and girls 54 percent (Table III). The variation in the proportion of the two sexes in the different subgroups is largely due to chance, as in larger subgroups the sexes are more or less equally represented.

As to age at time of onset of deafness, it was congenital or probably congenital in 66 percent; it was gradual in its onset in 8 percent; it occurred under one year of age in 5 percent; between

TABLE III.—SEX DISTRIBUTION ACCORDING TO ETIOLOGICAL CLASSIFICATION

<i>Cause of deafness</i>	<i>Boys</i>	<i>Girls</i>	<i>Total</i>
Congenital .....	18	32	50
Probably congenital .....	8	8	16
Cerebrospinal meningitis .....	11	3	14
Disease of ear, nose and throat...	5	7	12
Toxic neuritis .....	4	4	8
Total .....	46	54	100

the ages of one and two years in 10 percent; between the ages of three and five years in 5 percent; between the ages of six and eight years in 2 percent; and between the ages of nine and eleven in 4 percent (Table IV.).

TABLE IV.—AGE AT ONSET OF DEAFNESS ACCORDING TO ETIOLOGICAL CLASSIFICATION

<i>Cause of deafness</i>	<i>At birth</i>	<i>Probably at birth</i>	<i>Gradual onset; time uncertain</i>	<i>Age in years when time is known</i>					<i>Total</i>
				<i>Under 1</i>	<i>1 to 2</i>	<i>3 to 5</i>	<i>6 to 8</i>	<i>9 to 11</i>	
Congenital .....	50	..	..	..	..	..	..	..	50
Probably congenital .....	..	16	..	..	..	..	..	..	16
Cerebrospinal meningitis...	..	..	..	3	7	..	2	2	14
Disease of ear, nose and throat .....	..	..	8	2	..	1	..	1	12
Toxic neuritis .....	..	..	..	..	3	4	..	1	8
Total .....	50	16	8	5	10	5	2	4	100

The audiometric tests by air and bone conduction, on which the study is based, were made on our newly modified and amplified audiometer, in a sound-proof laboratory, and during a morning period to eliminate the fatigue element and the effects of auditory stimulation to the greatest possible extent. The signal button was used by the pupil, and all possible distraction eliminated; at the same time nervous strain was reduced to a minimum, the pupil being instructed to relax and rest his elbows on the arms of the chair in which he was sitting. In short, the tests were carried out in every possible way as controlled physiological investigations. The majority

of the pupils have taken at least two audiometer tests each year for the past four years; those who entered Clarke School during 1934 were given a sufficient number of tests prior to the one recorded for this study to eliminate any possible practice factor from the threshold values.

We are greatly indebted to Dr. Harvey Fletcher of the Bell Telephone Laboratories for devising and supervising the changes in our original 2-A audiometer. By means of these modifications, the intensity range has been increased 25 decibels for each pitch, and readings may be made at quarter-octave intervals from 64 to 8,192 d. v. and at two quarter-octave intervals above 8,192 d. v. With our amplified and modified audiometer, it is now possible to explore the entire auditory field of the deaf child. In the present study, readings have been made at octave intervals from 64 to 8,192 d. v., inclusive, and when necessary, the greatest possible intensity has been given the pupil. This means that we are now able to obtain a hearing curve from even the most profoundly deaf individuals, many of whom gave no response on the original 2-A. The added intensity range is illustrated in Chart 1, the dots indicating the limits

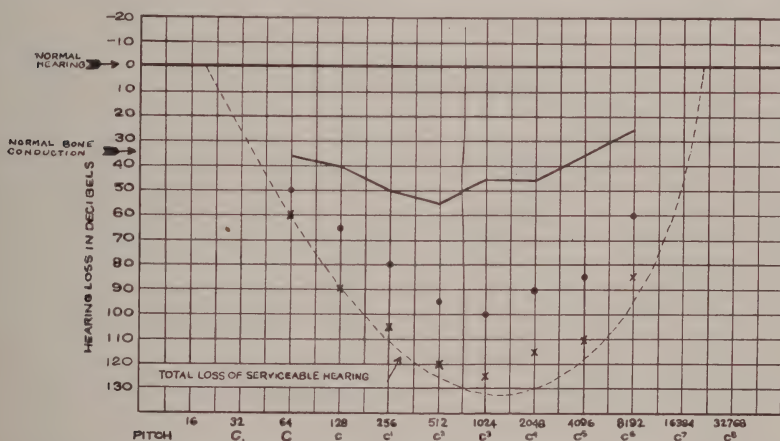
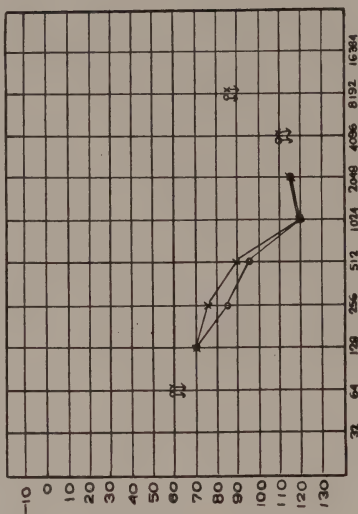


CHART 1. Audiogram showing intensity limits of the original 2-A audiometer by dots, of the amplified 2-A audiometer by crosses.

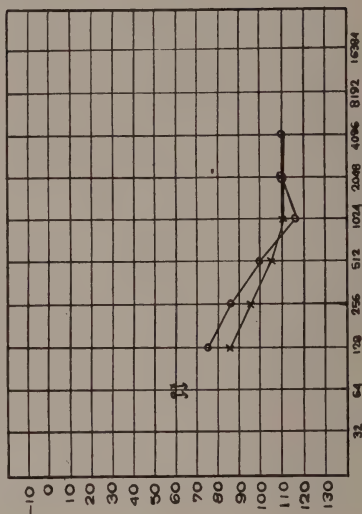
of intensity of the original 2-A, and the crosses the limits of intensity of our amplified 2-A. Chart 2 illustrates the advantages of the amplified audiometer in obtaining complete hearing curves from profoundly deaf individuals.

Hearing by bone conduction has been tested on the audiometer by means of the Western Electric bone conduction receiver placed over the mastoid region.

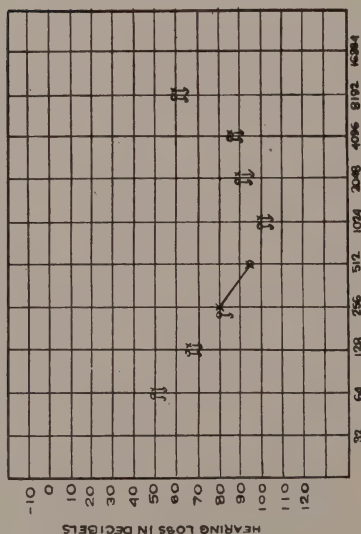
PUPIL 1. AMPLIFIED 2-A AUDIOMETER



PUPIL 2.



PUPIL 1 2-A AUDIOMETER



PUPIL 2.

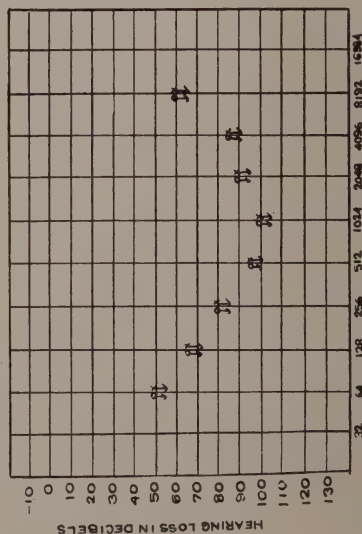


CHART 2. Audiograms showing advantages of amplified 2-A audiometer.



## STATISTICAL ANALYSIS\*

## A. AIR CONDUCTION

In the study of air conduction curves, we have sought to analyze the data in two ways: (1) the trend of the entire group, represented by the mean curve and the frequency distribution at each pitch; and (2) the trend for the four main etiological subgroups, represented by their respective mean curves and frequency distribution at each pitch.

In Chart 3, the mean curve for air conduction for the entire group has been plotted, with the frequency distribution at each pitch. It is a gently descending curve from 64 d. v. to 4,096 d. v. inclusive, with a very slight rise at 8,192 d. v. The shape of the curve itself is representative, but the scatter around the means is so great that the precise level is probably not established. In fact, the frequency distribution at each pitch is so very wide that it is evident at a glance that we are dealing with a far from homogeneous group in a school for the deaf.

In Chart 4, the mean curves for air conduction for the four largest etiological subgroups have been plotted. On the same chart, for comparative purposes, will be seen an average curve for air conduction obtained by testing a group of sixty normal individuals of approximately the same age range. It will be noted that the mean curve for the ear, nose and throat group is considerably higher than those for the other deaf groups; it descends gradually to 1,024 d. v., then remains horizontal except for a slight rise at 8,192 d. v. The mean curves for the congenital and probably congenital groups very nearly parallel each other, the mean curve for the probably congenital group being slightly higher until 4,096 d. v. when it reaches a slightly lower point, but it again crosses the mean curve for the congenital group and once more reaches a slightly higher point at 8,192 d. v. Both the curves descend through 4,096 d. v. and show a rather abrupt rise between 4,096 and 8,192 d. v. The mean curve for the meningitis group is by far the lowest of the four; it descends gradually to 1,024 d. v. and at 4,096 and 8,192 d. v. it very nearly coincides with the line of total loss of serviceable hearing, meaning that at the two higher pitches the group have no residual hearing and at all points the percentage of residual hearing is very small.

In Table V the constants of the frequency distributions are given for the entire group and the etiological subgroups in the form of

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\* We wish to acknowledge the advice of Dr. E. M. Jellinek, Director of the Biometric Department at the Worcester State Hospital, on the statistical methods used in this study.

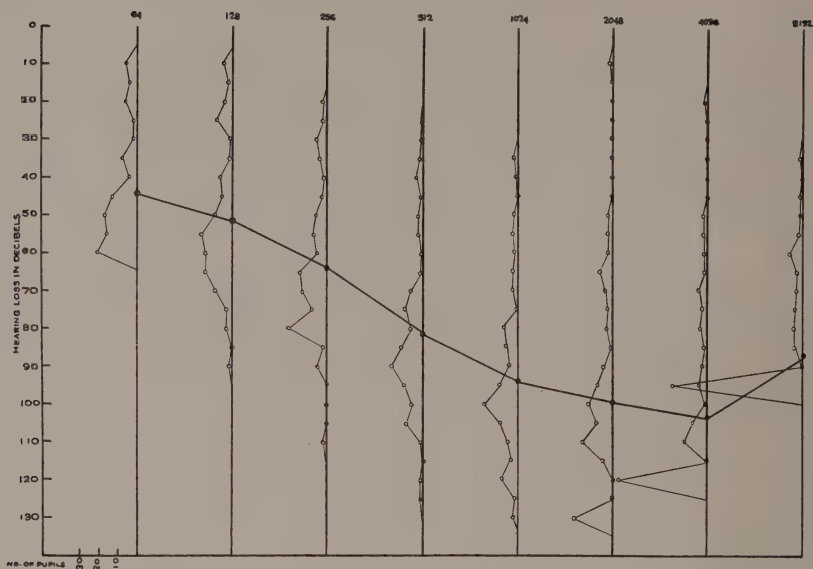


CHART 3. Mean hearing curve by air conduction for 100 pupils, with frequency distribution at each pitch.

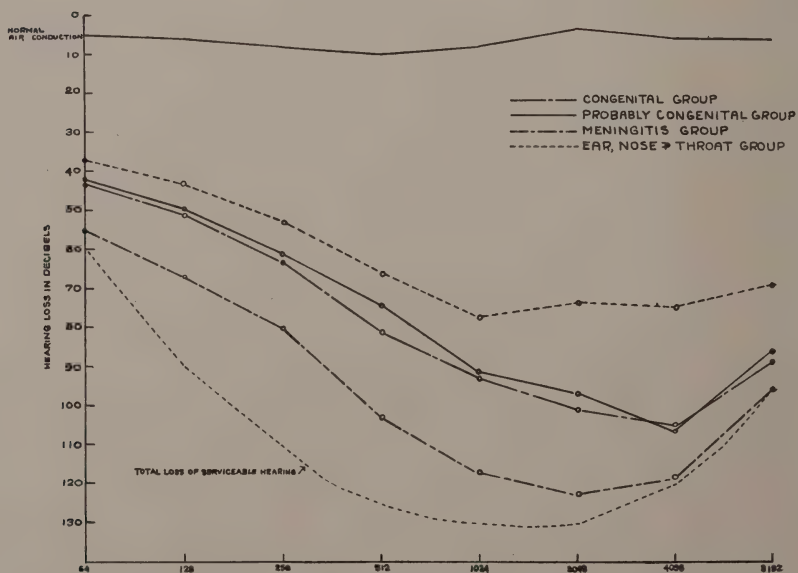


CHART 4. Mean hearing curves by air conduction for principal etiological subgroups with mean hearing curve for group of sixty normal individuals.

TABLE V.—CONSTANTS OF FREQUENCY DISTRIBUTION, INCLUDING MEAN, MINIMUM AND MAXIMUM READINGS AT EACH PITCH, BY AIR CONDUCTION, FOR THE ENTIRE GROUP AND THE ETIOLOGICAL SUBGROUPS.

Cause of deafness	No. of pupils	64 d. v.			128 d. v.			256 d. v.			512 d. v.		
		Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.
Congenital .....	50	43	10	60	51	15	80	63	30	90	81	35	105
Probably congenital....	16	43	10	60	50	10	75	61	20	85	75	30	95
Cerebrospinal meningitis	14	55	45	60	66	55	90	80	65	110	103	90	125
Disease of ear, nose and throat .....	12	37	10	60	42	10	80	53	20	90	65	25	100
Toxic neuritis .....	8	39	20	55	44	25	65	58	45	70	76	55	105
Entire group .....	100	44	10	60	51	10	90	64	20	110	81	25	125

TABLE V.—Concluded

Cause of deafness	No. of pupils	1,024 d. v.			2,048 d. v.			4,096 d. v.			8,192 d. v.		
		Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.
Congenital .....	50	93	35	120	101	50	130	105	55	120	89	50	95
Probably congenital....	16	92	55	120	97	65	130	107	70	120	87	60	95
Cerebrospinal meningitis	14	118	95	130	122	95	130	117	110	120	95	95	95
Disease of ear, nose and throat .....	12	77	35	115	74	10	130	75	20	120	69	35	95
Toxic neuritis .....	8	88	60	120	94	60	130	101	70	120	85	60	95
Entire group .....	100	94	35	130	99	10	130	103	20	120	87	35	95

the mean, minimum and maximum readings at each pitch. In order that we may compare not only the central tendency, but also the relative variability of these groups, the mean, standard deviation and coefficient of variation at the eight pitches have been computed for each group (Table VI), excepting the toxic group which was too small for such computations. The standard deviation is a measure of the "dispersion" or scatter of the individual readings from the mean or average reading at each pitch. The coefficient of variation is obtained by dividing the standard deviation by the mean and multi-

plying by 100 in order to estimate the relative variability at different pitches and in the different groups.

The central tendency, as shown by the means in Tables V and VI, is similar for the whole group and for the congenital and probably congenital subgroups, as was demonstrated graphically in Charts 3 and 4. The central tendency for the ear, nose and throat group and for the meningitis group differ as described above.

By comparing the coefficients of variation for the different pitches, both in the entire group and in the subgroups, it will be found that we have the widest scatter from the mean at 128 d. v., with the single exception of the congenital group in which there is the widest scatter at 64 d. v. The next widest scatter for all groups is at 64 d. v. All groups grow progressively more homogeneous from 256 to 1,024 d. v., and are most homogeneous at 8,192 d. v. In short, the scatter for all groups is widest for the lower tones and decreases progressively through the higher frequencies, as one would expect. In general for the entire group, the mean hearing loss tends to increase from the low to the high tones, and the coefficient of variation tends to decrease, meaning that, in general, the group has more hearing for the lower frequencies than for the higher, but that there is a much greater variation between individual readings for the lower frequencies than for the higher. Two exceptions should be noted: the coefficient of variation reaches a strikingly low point at 256 d. v. in the meningitis group and at 1,024 in the congenital group, meaning that at these two points the groups are for some reason strikingly homogeneous.

By referring again to Table VI, it is evident that the degree of variability of the entire group, the congenital group and the probably congenital group is very similar, the coefficients of variation ranging respectively from 35 to 16, 32 to 12, and 38 to 10.

By comparing the coefficients of variation for the subgroups, it is at once apparent that we have the widest scatter in the ear, nose and throat group, the coefficients for the eight pitches tested ranging from 59 to 29. This is as one would expect on the basis of the knowledge which we have concerning the after-effects of disease of the ear, nose and throat. In the group are individuals with chronic suppurative otitis media of long duration; in some the damage to the auditory apparatus has been very great, in others comparatively slight. The same is true of those who have had mastoiditis; some belong to the most profoundly deaf group and in some the damage is so slight that their graphs are at a high level and almost horizontal. Hence, the group in which deafness is due to primary disease of



TABLE VI.—MEAN, STANDARD DEVIATION AND COEFFICIENT OF VARIATION AT EACH PITCH, BY AIR CONDUCTION, FOR THE ENTIRE GROUP AND THE ETIOLOGICAL SUBGROUPS. S. D. INDICATES STANDARD DEVIATION AND V. INDICATES COEFFICIENT OF VARIATION.

Cause of deafness	No. of pupils	64 d. v.			128 d. v.			256 d. v.			512 d. v.		
		Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.
Congenital .....	50	43.4	14.0	32.3	51.3	16.1	31.3	63.3	15.9	25.2	80.9	16.8	20.7
Probably congenital .....	16	42.8	15.7	36.7	50.0	19.3	38.6	61.2	20.2	32.9	74.7	19.2	25.8
Cerebrospinal meningitis....	14	55.4	6.1	10.9	66.1	9.1	13.8	80.4	1.3	1.6	103.2	9.4	9.1
Disease of ear, nose and throat .....	12	37.1	21.8	58.9	42.1	24.9	59.1	52.9	24.9	47.0	65.4	25.5	39.0
Toxic neuritis .....	8	38.7	...	...	43.7	...	...	58.1	...	...	76.2	...	...
Entire group .....	100	43.9	14.7	33.6	51.5	18.2	35.5	63.7	18.1	28.5	80.8	20.4	25.2

TABLE VI.—*Concluded*

Cause of deafness	No. of pupils	1,024 d. v.			2,048 d. v.			4,096 d. v.			8,192 d. v.		
		Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.
Congenital .....	50	93.1	7.9	8.5	100.7	19.3	19.2	105.0	17.9	17.1	88.8	11.0	12.4
Probably congenital .....	16	91.6	13.9	15.2	96.9	18.1	18.7	106.9	11.4	10.7	86.6	12.5	14.5
Cerebrospinal meningitis..	14	117.9	10.1	8.6	122.1	13.1	10.7	117.1	7.2	6.2	95.0	...	...
Disease of ear, nose and throat .....	12	76.7	28.8	37.6	74.2	28.5	38.5	75.4	31.1	41.3	68.7	20.4	29.7
Toxic neuritis .....	8	88.1	...	...	94.3	...	...	100.6	...	...	85.0	...	...
Entire group .....	100	93.9	18.1	19.2	99.4	23.8	23.9	103.1	21.2	20.5	86.6	14.2	16.4

the ears, nose and throat is a far from homogeneous one, the graphs varying both in character and level, as is readily demonstrated by the mean curve, the frequency distribution table, and the coefficients of variation.

Once more referring to Table VI, it is apparent that there is a wide variation in the congenital and probably congenital groups, though somewhat less than in the ear, nose and throat group. This, again, is as one would expect. The fact that a child is born with

defective auditory function indicates nothing as to the particular part of the auditory apparatus which may be lacking or defective or the degree of the defective development. Hence, in the congenital and probably congenital groups, one may expect any type of graph at any level, the coefficients of variation ranging from 32 to 8 and 38 to 10, respectively. From our functional studies up to date, it seems evident that the congenital and probably congenital groups are in no sense of the word homogeneous groups from the standpoint of underlying pathology and resulting functional impairment. In order eventually to resolve these groups into their homogeneous parts, detailed functional studies over a period of years on a large number of congenitally deaf individuals must be linked up to detailed postmortem studies. We hope this will come as the result of a combination of research which is already under way in the various centers. It is apparent from the character of the mean curves and the frequency distribution that the congenital group and the probably congenital group are not essentially different. In future analyses, these will probably be carried as one group.

Finally, we come to the group in which deafness is due to cerebrospinal meningitis, which is, from the standpoint of etiology, the most homogeneous group in a school for the deaf. As is readily seen in Table V, the variation is strikingly less than in any other group, the coefficients of variation ranging from 13 to 0. The child is always profoundly deaf, as is evident from the mean curve.

Since our analysis showed that the means for the right, left, better and poorer ears were very similar in all groups with the exception of the ear, nose and throat group, we have based our analyses on the readings for the right ear throughout this study. In Chart 5 these four mean curves have been plotted for the ear, nose and throat group in which the two ears are sometimes quite unequally affected. It will be seen that the means for the right and the left ears are very similar except at 8,192 d. v. when the mean for the right ear is higher, while the mean curve for the better ear is considerably higher than that for the poorer ear at all points.

We have sought to analyze the bone conduction data according to the same plan, namely: (1) the trend or central tendency of the entire group, represented by the mean curve and the frequency distribution at each pitch; and (2) the trend or central tendency of the four main etiological subgroups, represented by their respective mean curves and frequency distributions at each pitch. Other points will be brought out under a comparison between air and bone conduction data.

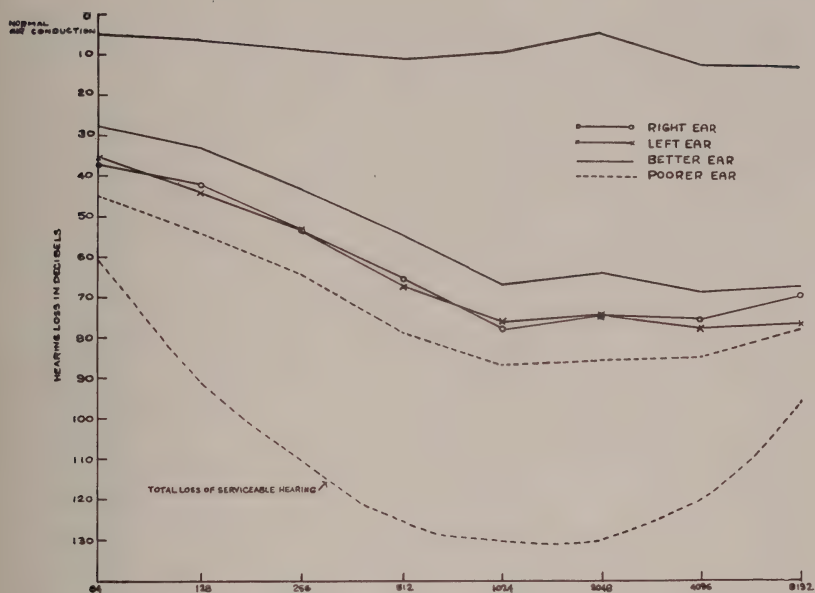


CHART 5. Mean curves by air conduction for right, left, better and poorer ears of the ear, nose and throat group.

#### B. BONE CONDUCTION

In Chart 6, the mean curve for the bone conduction readings for the entire group has been plotted, with the frequency distribution at each pitch. The curve descends gently to 128 d. v., then abruptly to 1,024 d. v., very slightly to 2,048 d. v., and from there rises steadily to 8,192 d. v. Beginning at 512 d. v., the mean rapidly approaches the line of total loss, and at the three higher frequencies, it is only very slightly above that line. For all practical purposes the mean is a descending curve, dropping off at 2,048 d. v. The trend of the mean curve is valid, being based on readings on 100 representative individuals. The level of the individual points is also reliable, as the scatter around the means is not great, as brought out by the smallness of the standard deviations and coefficients of variation in Table VIII.

In Chart 7, the mean curves for bone conduction for the four largest etiological subgroups have been plotted, and above them is the normal threshold for bone conduction. The order of the level of the mean curves is as it was in air conduction (Chart 4); the ear, nose and throat group occupies the highest level; the probably congenital, the congenital, and the meningitis groups follow in the same order. However, the mean curves for bone conduction for these

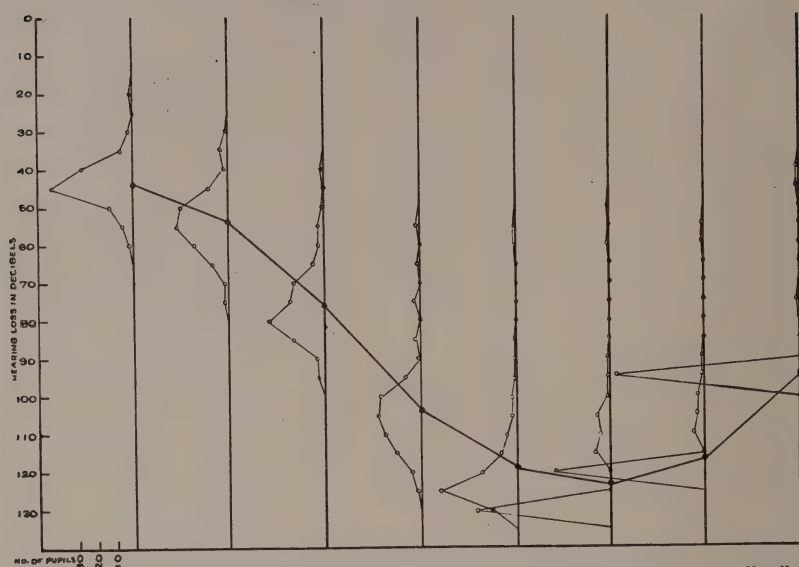


CHART 6. Mean hearing curve by bone conduction for 100 pupils with frequency distribution at each pitch.

groups are not nearly so widely separated as are those for air conduction. The mean curves for the upper three groups are close together until 256 d. v., when the means for the probably congenital and congenital groups descend more abruptly. The mean for the ear, nose and throat group descends to 1,024 d. v., then remains practically horizontal to 4,096 d. v., and ascends to 8,192 d. v. From 256 d. v. on the means for the probably congenital and congenital groups very closely parallel that for the meningitis group at only a slightly higher level. This is quite different from the relationship between the means for the same three groups by air conduction, as seen in Chart 4. The mean for the meningitis group by bone conduction descends abruptly, and coincides with the line of total loss for the three higher frequencies.

In Table VII, the constants of the frequency distributions by bone conduction are given for the entire group and for the etiological subgroups in the form of the mean, minimum and maximum readings at each pitch. In order that not only the central tendency, but also the relative variability, of these groups may be compared, we have computed in Table VIII, the mean, standard deviation and coefficient of variation at the eight pitches for each group as was done for the air conduction readings.



TABLE VII.—CONSTANTS OF FREQUENCY DISTRIBUTION, INCLUDING MEAN, MINIMUM AND MAXIMUM READINGS AT EACH PITCH, BY BONE CONDUCTION, FOR THE ENTIRE GROUP AND THE ETIOLOGICAL SUBGROUPS.

Cause of deafness	No. of pupils	64 d. v.			128 d. v.			256 d. v.			512 d. v.		
		Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Min.	Max.	Mean
Congenital .....	50	44	20	55	54	30	75	77	55	95	105	85	125
Probably congenital...	16	43	35	60	52	35	75	74	55	90	101	75	120
Cerebrospinal meningitis	14	48	40	60	59	50	65	81	70	95	110	100	120
Disease of ear, nose and throat .....	12	42	30	55	52	35	65	70	40	90	90	55	120
Toxic neuritis .....	8	44	40	50	53	40	65	79	60	85	106	75	120
Entire group .....	100	44	20	60	54	30	75	76	40	95	104	55	125

TABLE VII.—Concluded

Cause of deafness	No. of pupils	1,024 d. v.			2,048 d. v.			4,096 d. v.			8,192 d. v.		
		Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.
Congenital .....	50	121	90	130	125	90	130	117	100	120	95	75	95
Probably congenital...	16	119	110	125	124	105	130	119	110	120	95	95	95
Cerebrospinal meningitis	14	125	115	130	129	115	130	120	120	120	95	95	95
Disease of ear, nose and throat .....	12	103	55	125	102	50	130	101	55	120	86	40	95
Toxic neuritis .....	8	120	85	130	124	100	130	118	105	120	95	95	95
Entire group .....	100	119	55	130	123	50	130	116	55	120	94	40	95

For all groups, excepting the ear, nose and throat group, the widest scatter is at 64 or 128 d. v. and the smallest at 4,096 or 8,192 d. v., as indicated by the coefficients of variation. In the ear, nose and throat group, the widest scatter is at 512 and 2,048 d. v., where the coefficients are 23.4 and 23.6, and the least scatter at 64 d. v. where the coefficient is 15.7. The amount of variability at all pitches and in all groups is comparatively small, the highest coefficient of variation being 23.6 in the ear, nose and throat group, the lowest

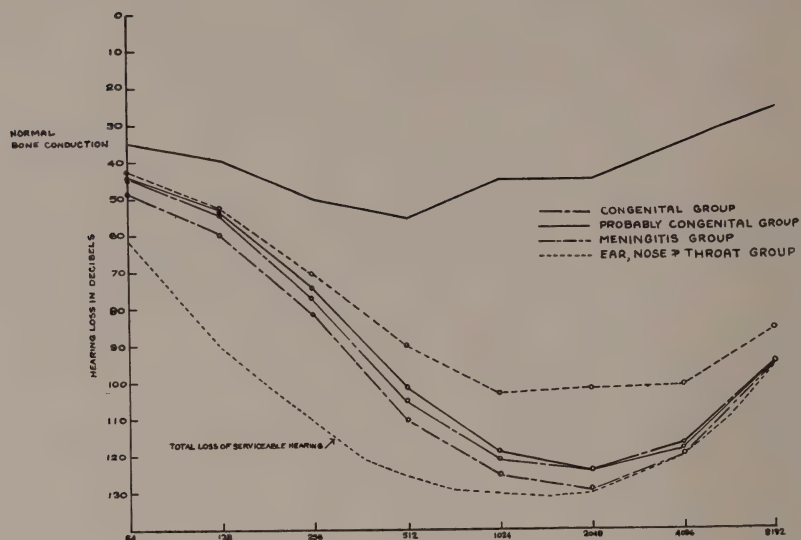


CHART 7. Mean hearing curves by bone conduction for principal etiological subgroups.

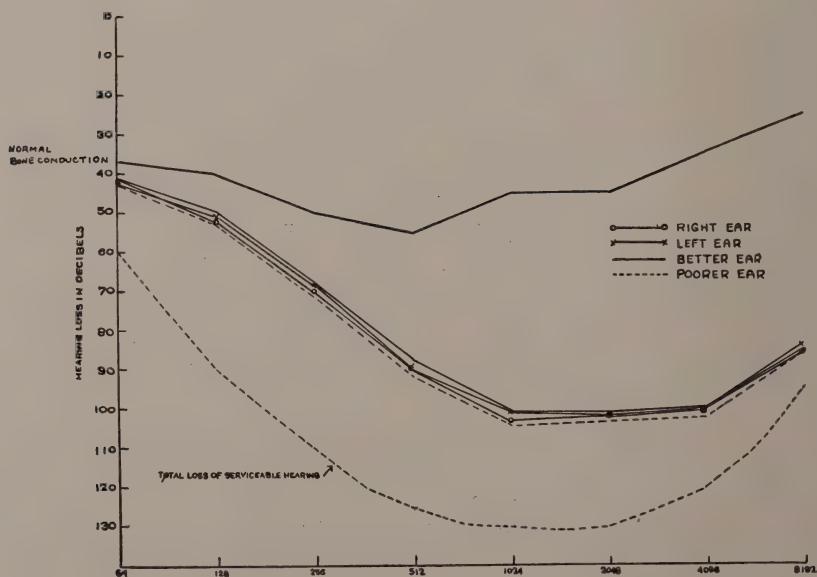


CHART 8. Mean hearing curves by bone conduction for the right, left, better and poorer ears of the ear, nose and throat group; better and poorer according to air conduction readings.

TABLE VIII.—MEAN, STANDARD DEVIATION AND COEFFICIENT OF VARIATION AT EACH PITCH, BY BONE CONDUCTION, FOR THE ENTIRE GROUP AND THE ETIOLOGICAL SUBGROUPS. S. D. INDICATES STANDARD DEVIATION; V. INDICATES COEFFICIENT OF VARIATION.

Cause of deafness	No. of pupils	64 d. v.			128 d. v.			256 d. v.			512 d. v.		
		Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.
Congenital .....	50	43.6	5.8	13.4	53.7	7.9	14.8	76.6	7.9	10.3	105.1	8.1	7.8
Probably congenital .....	16	43.1	6.8	15.8	51.9	8.6	16.7	74.4	8.6	11.6	101.3	13.6	13.5
Cerebrospinal meningitis ...	14	47.5	5.3	11.1	59.3	4.9	8.3	80.7	7.3	9.0	110.4	6.4	5.8
Disease of ear, nose and throat .....	12	41.7	6.6	15.7	51.7	9.6	18.7	70.0	14.9	21.2	90.0	21.0	23.4
Toxic neuritis .....	8	44.3	...	...	53.1	...	...	78.8	...	...	106.2	...	...
Entire group .....	100	43.9	6.1	13.8	53.9	8.2	15.3	76.2	9.5	12.5	103.5	12.4	12.0

TABLE VIII.—*Concluded*

Cause of deafness	No. of pupils	1,024 d. v.			2,048 d. v.			4,096 d. v.			8,192 d. v.		
		Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.	Mean	S. D.	V.
Congenital .....	50	120.6	5.4	4.4	124.9	9.7	7.7	117.1	6.8	5.8	94.6	2.8	3.0
Probably congenital .....	16	119.1	6.7	5.6	124.4	8.6	6.9	118.8	3.3	2.8	95.0	...	...
Cerebrospinal meningitis...	14	125.0	7.7	6.2	128.9	3.9	3.0	120.0	...	...	95.0	...	...
Disease of ear, nose and throat .....	12	103.3	22.4	21.7	102.0	24.1	23.6	101.3	22.0	21.8	86.3	19.7	21.8
Toxic neuritis .....	8	120.0	...	...	124.3	...	...	118.1	...	...	95.0	...	...
Entire group .....	100	118.9	11.4	9.6	122.6	14.1	11.5	116.0	8.0	6.9	93.8	7.6	8.1

being zero at 4,096 and 8,192 d. v. in the meningitis group, and at 8,192 d. v. in the probably congenital group.

As in air conduction, the degree of variability of the whole group, the congenital and probably congenital groups is very similar, the coefficients of variation ranging respectively from 15.3 to 6.9, 14.8 to 3.0, and 16.7 to 0.

The order of relative variability in the subgroups is similar to that for the air conduction readings, the ear, nose and throat group

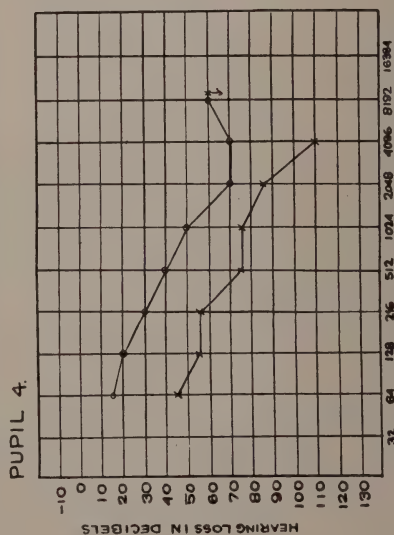
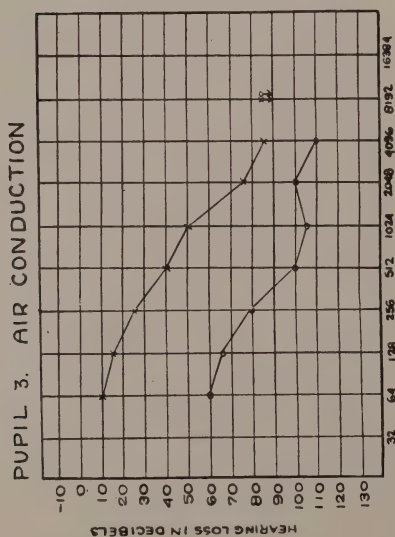
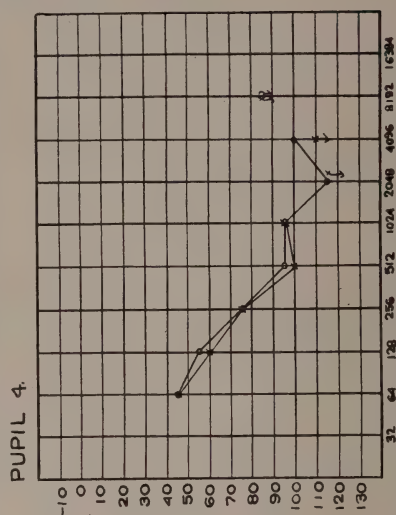
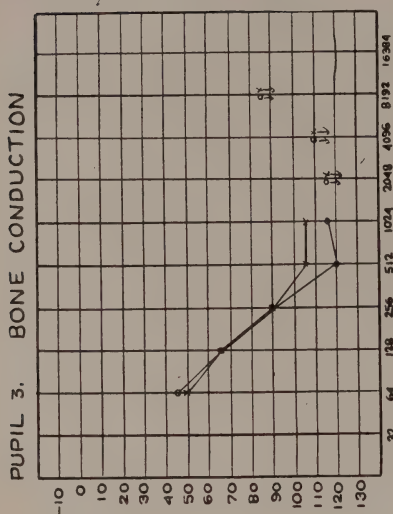


CHART 9. Individual audiogram showing dissimilar air conduction curves for the right and left ears and similar bone conduction curves.



showing the widest scatter and the meningitis group the smallest amount of scatter. However, the difference in variability between the subgroups is much less for bone conduction than for air conduction.

In the ear, nose and throat group unilateral differences do not seem as evident as by air conduction. In Chart 8, mean bone conduction curves have been plotted for the right, left, better and poorer ears in this group (better and poorer according to air conduction readings). The four mean curves lie very close together, the one for the poorer ear being at only a slightly lower level than that for the better ear. In Chart 9, individual audiograms are given for two pupils, one from the ear, nose and throat group and one from the congenital group. In both instances the air conduction curves for the right and left ears show marked differences, while the bone conduction curves for the two ears are very similar.

#### C. COMPARISON OF AIR AND BONE CONDUCTION DATA

In the comparison of air and bone conduction data, we have analyzed the material in four ways: (1) by comparing the general trend of the air conduction means for the whole group with that of the bone conduction means for the whole group; (2) by comparing the differences between the means for air and bone conduction for the entire deaf group with the differences between similar means for normal individuals; and by comparing differences between air conduction means for the entire deaf group and similar means for normal group with differences between bone conduction means for the entire deaf group and similar means for normal groups; (3) by comparing the general trend of air conduction means for the etiological subgroups with that of the bone conduction means for the same groups; (4) by attempting to determine whether a correlation exists between individual air conduction readings and individual bone conduction readings.

In Chart 10, the mean curve for air conduction for the entire group is represented by a line connected by circles, and that for bone conduction by a line connected by crosses. The trend of these two curves is as described in Sections A and B. They begin at the same level at 64 d. v., but the bone conduction curve descends more abruptly so that the difference between the level of the two curves increases steadily through 1,024 d. v., remains unchanged at 2,048 d. v., and decreases progressively to 8,192 d. v.

In the upper portion of Chart 10, curves for normal air and normal bone conduction have been plotted. In Table IX, differences

in decibels between means for air and bone conduction at each pitch for normal individuals have been compared with the differences between similar means for the entire deaf group. The differences

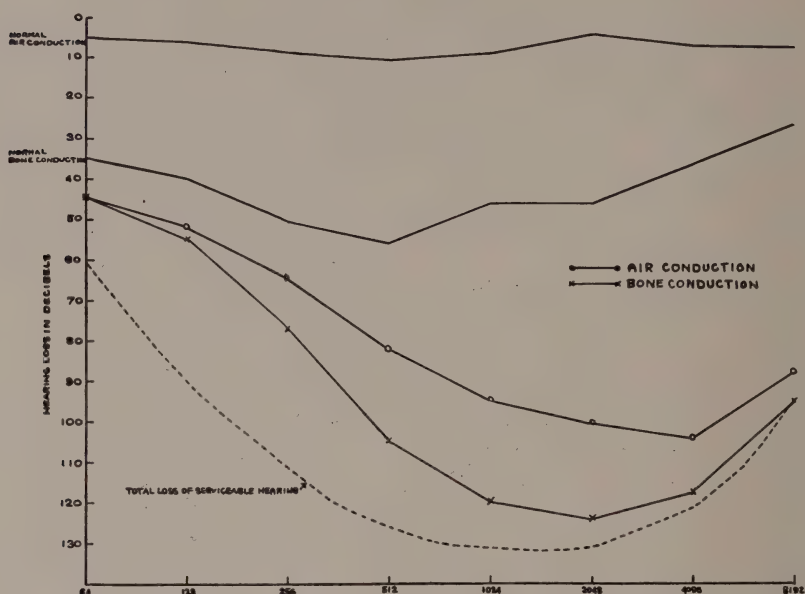


CHART 10. Mean hearing curves by air and bone conduction for the entire group.

between the air and bone conduction means for the deaf group are strikingly less at all pitches, but most markedly so at the three lower pitches.

Again in Table IX, differences in decibels between air conduction means for the entire deaf group and similar means for the normal group have been compared with differences between bone conduction means for the entire deaf group and similar means for a normal group. It is at once apparent both from the graphic presentation in Chart 10, and from the contrasting figures in Table IX, that the differences between the bone conduction means for the entire deaf group and normal bone conduction are considerably less at all pitches than similar differences between air conduction means for the deaf group and normal air conduction. This is most strikingly true for pitches 64 to 512 d. v., inclusive. At the four higher pitches the differences between the bone conduction means and normal bone conduction are slightly less. In other words, the average threshold for bone conduction in the deaf group is much nearer normal for the four lower frequencies than the average thresh-

TABLE IX.—COMPARISON OF DIFFERENCES BETWEEN THE MEANS FOR AIR AND BONE INDUCTION FOR THE ENTIRE DEAF GROUP WITH THE DIFFERENCES BETWEEN SIMILAR MEANS FOR NORMAL INDIVIDUALS; AND COMPARISON OF DIFFERENCES BETWEEN AIR CONDUCTION MEANS FOR ENTIRE DEAF GROUP AND SIMILAR MEANS FOR NORMAL GROUP WITH DIFFERENCES BETWEEN BONE CONDUCTION MEANS FOR ENTIRE DEAF GROUP AND SIMILAR MEANS FOR NORMAL GROUP.

<i>Pitch</i>	<i>Differences in decibels between air conduction and bone conduction means for normal group</i>	<i>Differences in decibels between air conduction and bone conduction means for entire deaf group</i>	<i>Differences in decibels between air conduction means for normal group and air conduction means for entire deaf group</i>	<i>Differences in decibels between bone conduction means for normal group and bone conduction means for entire deaf group</i>
64 d. v. ....	30	...	38.9	8.9
128 d. v. ....	34	2.4	45.5	13.9
256 d. v. ....	42	12.5	55.7	26.2
512 d. v. ....	45	22.7	70.8	48.5
1,024 d. v. ....	37	24.9	86.0	73.9
2,048 d. v. ....	42	23.2	96.4	77.6
4,096 d. v. ....	29	12.9	97.1	81.0
8,192 d. v. ....	19	7.2	80.6	68.8

hold for air conduction, while for the four higher frequencies this difference is less marked; it should be borne in mind, however, that the average bone conduction threshold for the three higher frequencies is almost coincident with the line of total loss, while the average air conduction threshold is considerably above such a line. The latter point has practical significance from an educational standpoint because of the importance of these higher frequencies for the discrimination of speech sounds.

Turning to Chart XI, the differences between the mean curves for air and bone conduction for the principal etiological subgroups are contrasted. The differences for the congenital and probably congenital groups are very similar to those described for the entire group. In the ear, nose and throat group both curves are at a higher level, and the difference between the two thresholds is greater at 64 d. v. and at 8,192 d. v. than in the groups just mentioned. In the meningitis group, both curves are very low, and the difference between the two thresholds is so small as to be almost negligible; at the two lower pitches the bone conduction threshold is slightly higher than that for air conduction; at the other frequencies the air conduction

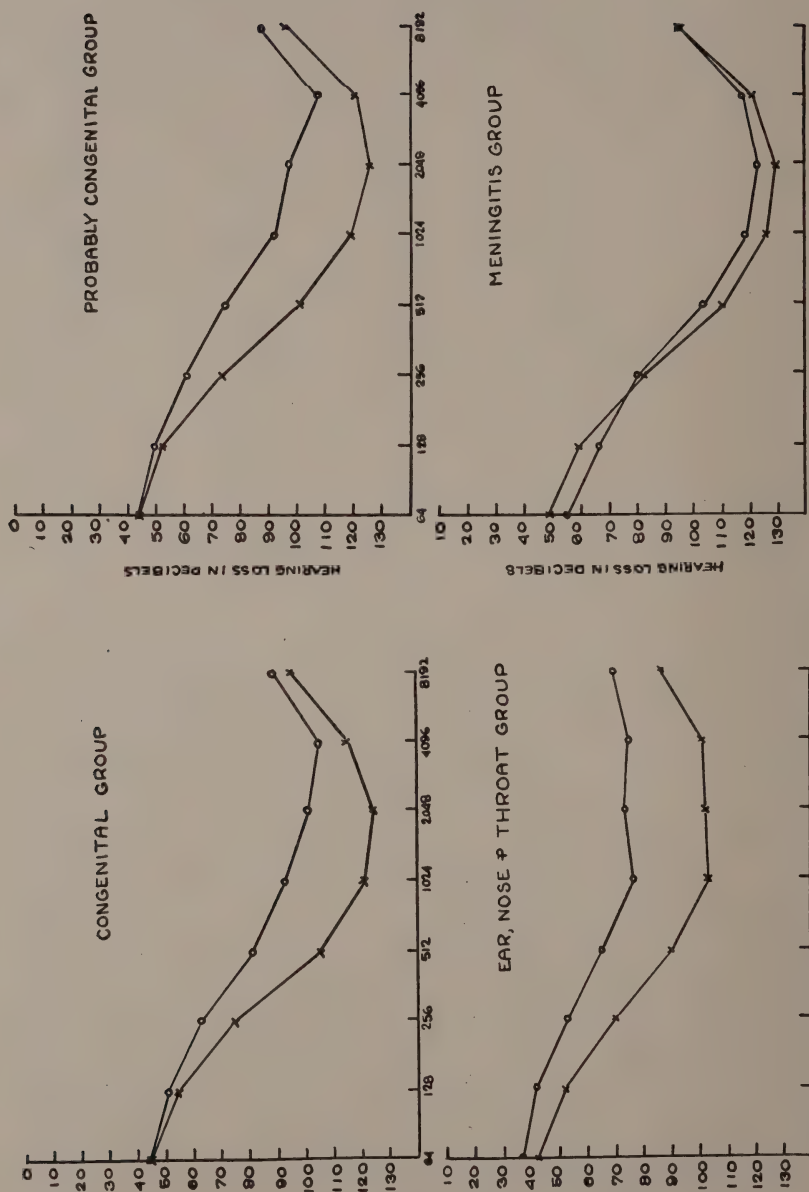
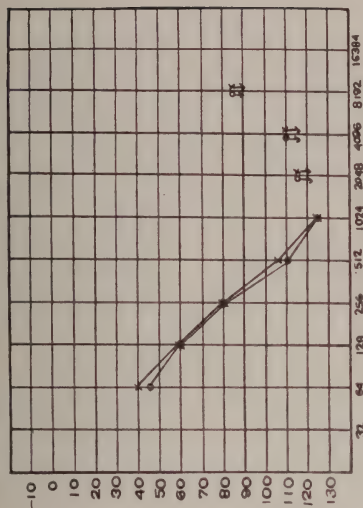


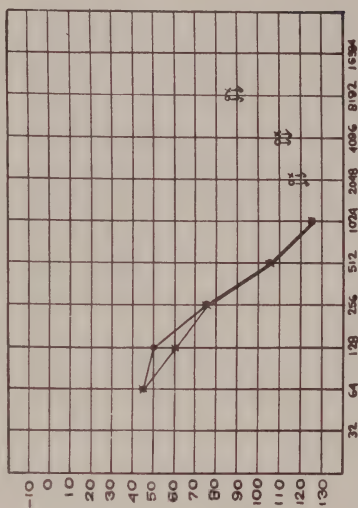
CHART 11. Mean hearing curves by air and bone conduction for the four principal subgroups.



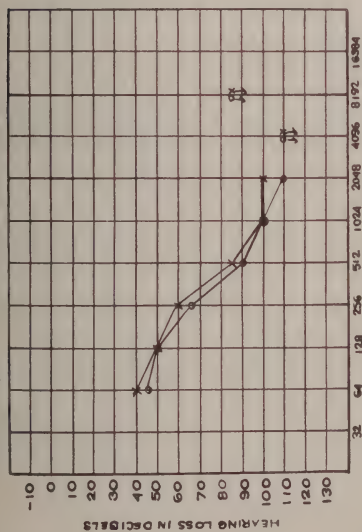
PUPIL 5. BONE CONDUCTION



PUPIL 6.



PUPIL 5. AIR CONDUCTION



PUPIL 6.

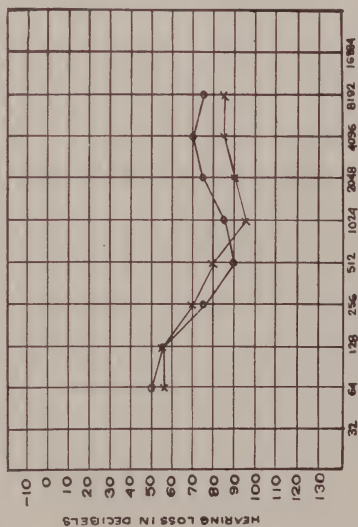


CHART 12. Individual audiograms showing dissimilar air conduction curves and similar bone conduction curves. Pupils 5 and 6 from congenital group.

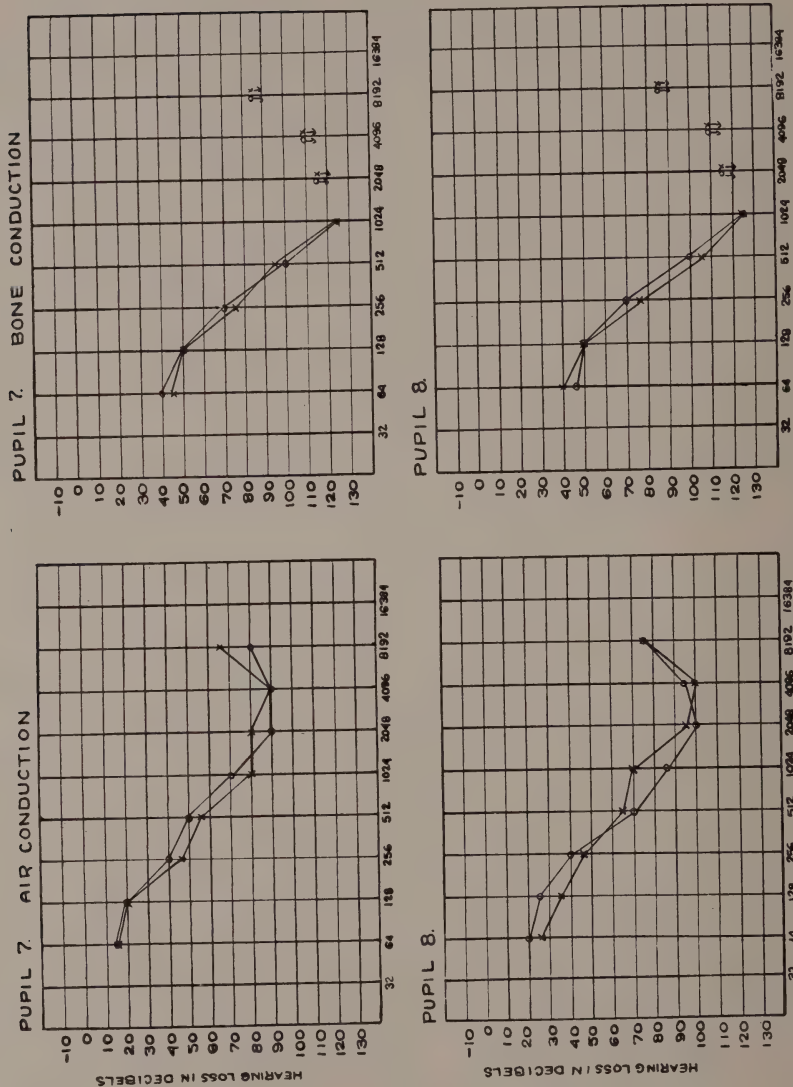


CHART 13. Individual audiograms showing similar air conduction curves and similar bone conduction curves.

threshold is slightly higher, except at 8,192 d. v. where both means indicate a total loss.

By referring to the frequency distribution in Charts 3 and 6, and to the coefficients of variation in Tables VI and VIII, it is at once apparent that the amount of variability at all pitches and for all groups is much less for the bone conduction readings than for air conduction, the differences being least marked in the meningitis group. In other words, both the entire group and the subgroups, with the exception of the meningitis group, are much more homogeneous from the standpoint of bone conduction than from that of air conduction. The variation in the meningitis group is very small by both air and bone conduction.

In order to determine whether a positive correlation exists between air conduction and bone conduction thresholds in a group of deaf pupils, scatter diagrams were made from the individual readings. It was found that there was no high, positive correlation. On the whole, the correlations were too poor to make it possible to predict either bone conduction from air conduction or vice versa. Pupils presenting similar air conduction curves may present like or unlike bone conduction curves and vice versa, as brought out in Charts 12 and 13.

#### COMMENTS

Inasmuch as these researches are being made in one of the oldest schools for the deaf in the country and one which has, since its founding, been a leader in this special field of education, we should like to mention very briefly the possible bearing of such findings upon the educational program for the deaf and partially deaf child. Through the greater delicacy of audiometers and hearing aids, the "hearing" of the "deaf" child is becoming a subject of increasing importance both from the scientific and the educational standpoint. The majority of children with auditory defects will unquestionably in the future be taught through the eye and the ear simultaneously, and their early speech-language development and future education will proceed more nearly along normal lines. But to bring this about most satisfactorily, science and education must proceed together.

In the analysis of the air conduction curves (Chart 3 and Tables V and VI), one is at once impressed with the lack of homogeneity in an unselected group of deaf pupils. With the increased emphasis on the use of residual hearing in the education of the deaf child, an educational classification on a scientific basis, either for school or for classes within the individual school, would seem to be of paramount importance. It is hoped that such detailed studies of auditory func-

tion in deaf pupils will stress anew this need; and that otologists and research workers will be able to cooperate with boards of education and schools for the deaf to this end. Can we not in this way make the most fundamental scientific contribution to the future welfare of the child who is already so handicapped that he needs some form of special education?

The data concerning the relation between air and bone conduction thresholds for the entire group, the subgroups and individuals should be helpful in the future in the selection of the type of amplification for various purposes. However, we wish to correlate these findings with the results of speech sound tests before we draw positive conclusions on these points.

#### SUMMARY

The study consists of a general survey of auditory function in 100 unselected pupils at The Clarke School for the Deaf, based on a statistical analysis of audiometric readings, by air and bone conduction, made with the amplified 2-A audiometer, which renders possible the exploration of practically the entire auditory field in the deaf child. The means, constants of frequency distribution, standard deviations, and coefficients of variation have been computed for the entire group and the main etiological subgroups by both air and bone conduction, and the air and bone conduction data for the entire group and the subgroups have been compared.

The trend of the entire group and the subgroups by both air and bone conduction may be seen in the accompanying charts and tables.

The most striking finding from the air conduction data was the great lack of homogeneity for all groups, with the exception of the meningitis group. For the entire group the mean hearing loss tends to increase from the low to the high frequencies and the coefficients of variation tend to decrease. This lack of homogeneity stresses anew the need of an educational classification on a scientific basis for schools or classes within the individual schools, if the deaf child is to receive the maximum benefit from the use of his residual hearing.

The most striking finding from the bone conduction data was the low level of the average threshold for the higher frequencies, the mean curve being almost coincident with line of total loss of serviceable hearing, while for the lower frequencies the means were surprisingly high.



The air conduction findings for the entire group and the etiological subgroups differ from those for bone conduction in the following ways:

(1) The mean air and bone conduction curves for the entire group begin at the same level and descend, but the mean bone conduction curve descends more abruptly. While the average threshold for bone conduction in the deaf group is nearer normal for the lower frequencies than the average threshold for air conduction, it must be borne in mind that at the three higher frequencies it is almost coincident with the line of total loss, while the mean air conduction curve is considerably above such a line. The latter difference has significance from an educational standpoint because of the importance of these higher frequencies for the discrimination of speech sounds.

(2) The order of the level of the mean curves for the etiological subgroups is similar for both air and bone conduction, the curve for the ear, nose and throat group occupying the highest level, the probably congenital, the congenital and the meningitis groups following in the order given, but the mean curves for bone conduction for these groups are not nearly so widely separated, the means for the congenital and probably congenital groups being at only a slightly higher level than the means for the meningitis group.

(3) The entire group and the subgroups show much less variability at all pitches by bone conduction than by air conduction.

(4) Unilateral differences in the ear, nose and throat group are not so marked by bone conduction as they frequently are by air conduction.

# STUDY OF AUDITORY FUNCTION IN 100 PUPILS AT THE CLARKE SCHOOL FOR THE DEAF

By RUTH P. GUILDER AND LOUISE A. HOPKINS

## OTOLOGICAL COMMENT

By GORDON BERRY, M.D.

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### I. NATIONAL STUDY OF DEAFNESS

American otologists are scrutinizing deafness. The American Otological Society has sponsored the careful study of otosclerosis. The America Federation of Organizations for the Hard of Hearing has been encouraging a nation-wide search for incipient cases of impaired hearing in our public-school children. The National Research Council, working with our deaf-school children, conducted an intensive study into the physical cause of deafness. In his presidential address before the American Otological Society in 1931, Dr. D. Harold Walker stressed research in otology as one of the most "fascinating" of the existing fields. The research work since conducted by Doctors Wever and Bray in Princeton, by Doctors Crowe and Guild at Johns Hopkins, by Doctor Fowler and his son at Columbia, by Doctors Lurie and Davis at Harvard, by Doctor Wilson at Chicago, contributes increasingly to our as yet uncertain knowledge. McNally, Tait, Rowe, Drury, Jones, MacFarlan, Davenport, Bast, Anson, Culler and Bunch are all joining in the attack, and there are many more. It is an impressive list. Otology then, is embattled against deafness.

### II. CLARKE SCHOOL RESEARCH

It was in 1927 that a drive was made for an endowment for The Clarke School for the Deaf at Northampton, Mass., some of which was to be devoted to research. It was the resulting Coolidge Fund that has permitted the addition to the faculty of an earnest group of research experts, among which are numbered our guests, Doctor Guildler and Miss Hopkins. We expect many interesting contributions from these zealous investigators.

1. *Method Pursued.*—This contribution, though reviewing the hearing acuity of only a single and limited group, deals with a char-

acteristic and not selected group, considers it intensively and in detail, and employs a recently devised calibrated amplification which permits accurate estimates in certain pitch ranges where no measured acoustic response had been previously obtained. It tries to plot a characteristic curve for this type of deafness, a curve which may be considered basic and serving as a standard. These 100 tests are not enough to establish accurately and finally such a mean curve, but these figures conform so closely to figures obtained by these same investigators through four years of detailed measurements, and the coefficient of error has been so carefully estimated, and the averages have been so mathematically computed and plotted, that I venture to say that subsequent investigations will change but little the resulting mean, unless there is a considerable and unexpected change in the type of deaf child admitted. We may then, for the present, accept these curves as true for the average child with profound deafness.

2. *Etiological Findings.*—Before discussing these essential curves, permit me a reference to the etiological findings in this group and an approximate comparison with Doctor Shambaugh's monumental work from 1925 to 1928 (*Archives of Otolaryngology*, May, 1928) when he and his assistants examined and tabulated 3,120 deaf children, and later reported on 5,000 cases (TRANSACTIONS, American Laryngological, Rhinological and Otolological Society, 1933). Doctor Shambaugh listed 60 percent as congenitally deaf; The Clarke School group had 50 percent of congenital and 16 percent of probably congenital origin, a total of 66 percent. Doctor Shambaugh found an associated otitis media in about one-third of the 40 percent acquired deafness cases or roughly 13 percent of the total, but in relatively few (2.6 percent) did he consider the middle ear infection the cause of the deafness, the balance being due to a coexisting toxic neuritis. Doctor Guilder and Miss Hopkins found about the same proportion troubled with "ear, nose and throat disease," namely 12 percent, but under their terminology attached major significance to it as the cause of the deafness. Of the meningitis group, Doctor Shambaugh had 8 percent; and this series shows 14 percent. Of deafness following the acute infectious diseases, Doctor Shambaugh found about 16 percent; and this series shows 8 percent, which is listed under "toxic neuritis." Here again, the classification being different, the results cannot be expected to harmonize closely. In the 1933 report, Doctor Shambaugh contrasts these deaf children with 165 hard-of-hearing (not deaf) adults, where 70 percent were due to otosclerosis, 5 percent were senile nerve forms, 1 percent were congenital, and 12 percent were tubo-tympanic, an interesting but scarcely significant

repetition of the 12 percent incident of "ear, nose and throat diseases" in The Clark School group.

3. *A Consideration of the Charts.*—a. Accuracy of the charts. These investigators have presented many charts, some of which I am unable as yet to completely understand. Those who are accustomed to working with statistical tabulations, feel at home when discussing "standard deviation" and "coefficients of variation." Here, I am quite out of my depth. But I follow the argument closely enough to realize the painstaking care with which the element of error has been reduced to the minimum, and to feel assured that these valuable charts are as nearly accurate and reliable as human skill can make them. They deserve careful scrutiny.

b. The consistent drop in the upper tones. Chart 10 gives in abstract the entire story. Above are two lines established by actual test with this same apparatus, showing the air conduction and the bone conduction for 60 normal children of the same age range. Next comes the mean air conduction line of this series (indicated by circles) and then the mean bone conduction line (indicated by crosses). Below this is the dotted line showing the limits of hearing. Please note the definite and characteristic drop or slide-off in the upper tone range, both by air and by bone. This slant is even more noticeable in my office tests for my 2-A Western Electric audiometer does not give the extra 25 decibels for each pitch that this amplified instrument of Doctor Fletcher's does, and the child frequently shows no response in the upper half of the tone range. It should be noted that the 1-A audiometer offers extra intervals and greater amplification than the standard 2-A, but it impresses me as less accurate and less efficient for this type of case than this specially devised machine. Doctors Guilder and Fowler can speak more authoritatively here.

To return to the graphs, this slanting line in the lower tone range is consistently found in almost all of these deaf children. Note also the similarity between the air and the bone conduction curves, a similarity not so frequently found in our office tests of the adult hard of hearing. I am hoping that in the discussion Doctor Fowler will contrast for us his graphs on hard of hearing children.

c. Four illustrative cases. I have selected from my office records of four individual deaf children, pupils of the exact type we are considering.

*Case 1* was a patient fourteen years old. No deafness in the family. Scarlet fever in infancy. A good clear voice, which is significant in these cases. Had been at Clarke School. Heard conversation ad oram on the left, possibly a shout on the right. Showed slanting graph, both by air and by bone.



*Case 2* showed a graph that was barely caught on the non-amplified 2-A audiometer. Aged nine. One uncle, hard of hearing in middle life, whose child (a cousin of the patient) was born deaf. This patient had double pneumonia at eleven months and whooping cough as a baby. She heard by air conduction in both ears on the 512 pitch only and by bone conduction in the left ear at the 256 pitch only.

*Cases 3 and 4*, the patients were sister and brother, aged eight and eleven, respectively. Both were "born deaf." They had first cousins who were both deaf and blind. I think of *Case 4* as quite typical if the non-amplified 2-A audiometer is used for the test. Here again I found the slanting line, and in the lower tone range.

d. The pathology suggested by these graphs. A reference to Dr. Stacy R. Guild's discussion on the Interpretation of Audiograms before the American Otological Society in 1933, is relevant. He makes a plea for audiometric readings as our most accurate means of testing for otosclerosis but reminds us that other tests should be made where a careful diagnosis is sought. He adds: "Ears whose audiograms indicate that all high tones are poorly or not heard, while the low tones are well heard, have shown the most consistent pathology of any group thus far studied. The essential lesions are in the basal turn of the cochlea, and consist of partial or complete atrophy of portions of the organ of Corti, the nerve fibers in the osseous spiral lamina, the cells of the spiral ganglion, or all of these elements."

e. The etiological groupings. Chart 4 shows an interesting similarity in the air conduction curves of the four principal etiological groups: the ear, nose and throat group above; the congenital and probably congenital coinciding; and the meningitis group below, closely approaching the total deafness line in the higher pitches. Chart 7 shows an even more striking parallelism in the bone conduction curves. Chart 11 contrasts the air (above) with the bone (below) in the four etiological groups. The more extreme the deafness, the more nearly does the air conduction approach the bone conduction. Here lies an element of error which these investigators have had to guard carefully against. It has been pointed out by others that with extreme sound magnification, air conduction approaches a tactile phenomenon and so simulates bone conduction. Dr. Douglas MacFarlan emphasized this tactile element in a paper before the American Otological Society in 1933 and said it was more likely to trouble in children. This was touched upon by Dr. E. P. Fowler at the same meeting when he gave his masterly analysis of 126 cases of otosclerosis. The tactile hazard he finds is in the lower half of the tone scale.

## III. CONCLUSIONS

In concluding permit me to emphasize five points. First, most of the children in our schools for the deaf have some hearing. Second, the downward trend of all these curves is characteristic of this type of deafness. Third, this is, I think, the first careful series where bone-conduction tests have been done in this type of case. I personally find that the bone conduction offers important evidence. Increasingly in my office tests, though there are so many exceptions, in general I sense that a poor air conduction and a good bone conduction offers a better prognosis, than a somewhat better air conduction and a lower bone conduction. Relatively speaking, the air conduction seems to seek the bone conduction level of acuity, as time elapses. Others here may have noted this trend. Fourth, Doctor Guilder has touched on the influence such careful acoustic analyses may have on the educational program. This is important. These tests were inclusive and offer the mean between the extremely deaf and those with considerable hearing. There have been children with 50 percent hearing in our schools for the deaf because there was no other place to educate them. And until only recently these have been universally taught in just the same way as if they were totally deaf. Group ear-phones are now available for these partly deaf, and our institutions for these unfortunates are in process of reshaping their teaching methods. Out of it will perhaps come a regrouping of the children somewhat in accordance with their hearing capacity. See then how important such careful hearing tests promise to be in the future, and how essential will be such information as Doctor Guilder and Miss Hopkins are assembling.

## DISCUSSION

D. HAROLD WALKER, M.D., Boston, Mass.: The authors were fortunate in possessing an amplified audiometer, otherwise many of the tests could not have been made, especially those by bone conduction, in the higher pitches. In fact, my only doubt of the accuracy of this work, would be that the amplified instrument extends the range of testing too high—too near the line of total loss of serviceable hearing, and that certain positions in the high pitches were tactile sensations, rather than actual hearing, but the great similarity of all curves, both by air and by bone conduction, are so consistent that this criticism is rather precluded. We have, then, standard curves of air and bone conduction of totally deaf pupils above the age of nine years. How different these curves are from those of the usual office patient!

The work of these researchers ceases here. They have given us their results and no interpretation is attempted. If all this work is to be of use, we must know what conclusions to draw. Let us exclude the cases of meningitis, a

known cause. That leaves the congenital, the probably congenital, and the nose and throat group. At first, one might say that no nose and throat case ever gave such a curve as shown, and there must be some explanation why this curve follows the curves of the two other groups. The explanation may be in the fact that the middle ear changes have occurred immediately after birth, or even prenatally, and hence the objective findings should be eliminated. In other words, this 12 percent should also be considered as probably congenital. We have three possible explanations of the cause of deafness in these pupils: first, embryonic defects in the organ of Corti, or in the neural content of the eighth nerve or its ramifications; second, otosclerosis; and third, a toxic neuritis. The diagnosis of otosclerosis is still, to me, a difficult one to make, so that a toxic neuritis may be the most likely cause; and I may add a fourth explanation, namely, atrophy from disuse, due to middle ear changes at birth or in very early life.

I have in mind two children, four-year-old twins, who were sent to me by the Speech Readers Guild, and who have been under the care of an expert pediatrician since birth. They were supposed to be congenitally deaf mutes. Examination showed intense retraction of the drum membrane. Under an anesthetic, small amounts of adenoid tissue were removed from each child, and the Eustachian tubes were catheterized, and bougies passed. One child is now able to speak certain words, and short sentences, and the other is beginning to appreciate the radio, and sound in general.

Important lessons may be drawn, namely, that the earlier and more thorough and more painstaking the examination of all deafened children, and their constant supervision by the otologist, then the greater realization of the prevalence of deafness in children by the general practitioner and the child specialist; and lastly, the necessity of constant effort to make use of what remnants of hearing exist, by means of the various instruments and also by lip reading.

In a paper written by Fritz Heider of The Clarke School, published in 1933, on "The Influence of the Epidemic of 1918 on Deafness," he speaks of the enormous increase in the number of children in the schools for the deaf, children who were born in the latter half of 1918, at the time of the epidemic of influenza. For example, the births of children registered in sixteen American schools for the deaf, previous to the epidemic, vary, month by month, from 24 to 56, but in the month of August, 1918, the number jumped to 103, in September 138, and in October 122, and then the next month back to 69, and in December to 48. Does this not show that there must have been a prenatal toxemia, which probably caused deafness in these cases? Inasmuch as the largest number of these births occurred in the extreme southern states where malaria is so very prevalent during the three months mentioned, the administration of quinin may have been an important factor.

Recent investigations on animals have shown that two-thirds of the cochlea can be destroyed and still the entire musical scale can be passed through the remaining whirl. I believe the time is coming when we can take advantage of the residual hearing, especially where the low tones are heard best, and gradually re-educate that portion of the cochlea to appreciate and transmit the higher tones.

Hitherto the fact that we have not made a diagnosis of petrositis concurrently with mastoiditis was because the signs and symptoms of mastoiditis

were dominant and that when a thorough mastoidectomy was done the changes in the deep cells surrounding the labyrinth subsided because of proper drainage and depletion and we never even considered them to be involved.

In retrospection I can think of several cases in which pain in the distribution of the fifth nerve was a predominant symptom but was never considered as anything but a symptom of mastoiditis which entirely subsided following the mastoidectomy. Today, I would, unhesitatingly, attribute that symptom to petrositis.

Unquestionably the treatment from an anatomical standpoint in suppurative petrositis and even non-suppurative petrositis is surgical, varying from a simple to the most radical procedure. The indications and type of operation I leave for others to discuss.

E. P. FOWLER, New York, N. Y.: This very careful study of the hearing in 100 pupils in the Clarke School for the Deaf presents numerous interesting facts for discussion. The amplified 2-A audiometer is about equal to the original 1-A audiometer in my possession except for lack of the 11,000 and 16,000 frequencies. It has one-quarter octave intervals instead of my one-third octave spacing.

The audiograms show general trends of curves for four different etiological groups all of which with the exception of the ear, nose and throat group showed for the mean curves similar forms and very severe deafness, more marked for the upper tones. Similar characteristics show in my own cases of very severe deafness and are to be expected, because the greater the nerve deafness the less the superimposed obstructive lesions can alter the form of the curve. This is shown clearly in the bone condition graphs (Chart 7) where for all but the ear, nose and throat group, the mean curves are nearly identical.

Statistical analysis of air conduction alone is of little value, because one finds all forms of air conduction curves in non-nerve lesions, many being typical of nerve deafness. Therefore, bone conduction measurements are necessary to assure a correct diagnosis.

Remember there may be and usually are present several co-existing lesions in the same ear. As the hearing becomes less and less more and more of these co-existing lesions are masked by the dominating cause of the deafness. This is most notable, therefore, in the congenital and meningitis groups and less noticeable in the ear, nose and throat group, because in the latter the nerve deafness does not dominate the picture to the same extent as in the former.

The meningitis curves so closely coincide with the total loss lines that practically these indicate a total loss of serviceable hearing. In these cases the so-called "residual hearing" is really more of a "hyperacute tactile sense" in the organ of hearing (located in the external auditory canal, the middle ear or the inner ear, the skin or even the periosteum, endosteum and dura).

As was to be expected the scattergrams show widest for the low tones, and especially in the ear, nose and throat group. All become more homogeneous as the scale is ascended largely because in the upper tones the average hearing losses are much more marked than in the lower tones. The great variability found in even the congenital groups gives hope that preventive measures may be found to counteract at least some of the lesions causing severe deafness.



The mean curves by bone conduction are not so widely separated as the air conduction curves chiefly because bone conduction shunts the testing sound around the obstructive lesions thus cutting out many causes of variability. Note that for all the groups the bone conduction curves are close together at 64, 128 and 256 cycles. Also it should be noted that the bone conduction measurements are always as good as and usually much better than the air conduction measurements. This is not apparent at first glance on the graphs because the writers have plotted the bone conduction curves using the irregular guide base line for bone conduction minimum audibility instead of the straight base line indicating normal hearing. In order to obtain a clear picture I have always maintained that the line representing normal hearing for air conduction should also represent normal hearing for bone conduction. I did not approve of inserting in the audiogram charts the line representing normal bone conduction because if this is used as a base, it makes the bone conduction appear on the chart 35 to 55 decibels lower than it really is, depending on the frequency, and it is apt to be misinterpreted unless one is accustomed to using two base lines at the same time, one (the straight one) for normal hearing by air conduction at one level, and the other (the crooked one) for normal hearing by bone conduction at a lower level.

The writers show the bone conduction mean curves for all but the ear, nose and throat group practically coinciding with the lines of total loss above 1,024 cycles whereas it really should be placed some 40 to 60 decibels higher than this. The first would indicate no serviceable hearing, the latter hearing at most no worse than an 80 db. loss. These two bone conduction graphs would indicate radically different methods of teaching speech, lip reading, etc.

One of the outstanding surprises of the hearing tests in this "fair sample" from the pupils of the Clarke School is the large amount of serviceable hearing possessed by many pupils. Students with serviceable hearing should not be in the classes for the deaf. They need for associates normal hearing children. This is understood by all teachers of the deaf and partially deaf. It is fundamental in the best teaching methods.

A word about the bone conduction measurements of a pair of ears. The reason the authors found bone conduction differences less evident than air conduction differences was, first, because bone conduction is more dependent upon the nerve function, and second, because the testers did not (and in many instances could not) mask the opposite ear while testing. If one ear is not properly masked the bone conduction will vary as between the two ears only 5 to 10 db., because sound is transmitted across the head to the opposite ear with an impedance of only 4 to 8 db.

The average threshold for bone conduction is so much nearer normal for the four lower tones, than is the case for air conduction that I wonder, whether such readings really signify measurements by bone conduction. May it not be that children who have been intensely trained especially to feel vibrations not only as sound but as a tactile sensation, are responding to tactile sensations and not to sound? I cannot avoid this conclusion in certain cases. Using the bone conduction receiver (or tuning fork) the low tones may be sensed by the finger tip at but little greater intensity than the threshold for minimum audibility by bone conduction, and for the lowest tones the tactile sense is even more sensitive than the sense of hearing by bone conduction.

# A REVIEW OF THE AURAL AND ORBITAL COMPLICATIONS OF SCARLET FEVER AT THE PHILADELPHIA HOSPITAL FOR CONTAGIOUS DISEASES FROM 1922 TO 1934

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Scarlet fever produces more cases of acute otitis media than any other specific disease in this country. A close second place is granted to measles. Some writers<sup>1</sup> would place measles above scarlet fever in their respective communities. This may be true, certainly both are prime producers of ear trouble. The writer holds the opinion that in this community the percentage of scarlatinal otitis which proceeds to surgical mastoiditis is substantially higher than of measles.

Acute suppurative otitis media is the most important complication of scarlet fever. If we may except lymphadenitis in general and serum reaction in those patients who receive serum, we may also safely say that acute otitis media is the most frequent, as well as the most important complication of scarlet fever.

In a previous report,<sup>2</sup> a review was given of the aural complications in scarlet fever at the Philadelphia Hospital for Contagious Diseases for the years 1922 to 1929, inclusive. It is the purpose here to bring the data up to, but excluding, 1934 in a consolidated report on otitis and to add a brief report on the orbital complications.

In this period of twelve years, 1922 to 1934, 29,298 scarlet fever patients were cared for at the hospital. Two thousand eight hundred and ninety-eight of them, or 9.9 percent, developed acute suppurative otitis media.

It is interesting to note that approximately one-third of the patients who developed a running ear had a bilateral affection, for among the 2,898 patients there were 4,100 running ears. Four hundred fifty-five of these ears, belonging to 363 patients, proceeded to surgical mastoiditis and were subjected to simple mastoidectomy.

The mortality rate for the mastoid patients was 9.6 percent. The total mortality rate for the 2,898 otitic patients was 3.7 percent.

The average number of days from operation to recovery, meaning a healed wound and a dry ear, was 47 for 313 unselected mastoid cases where the period could be determined from the records.

As regards myringotomy, these 4,100 ears may be divided into three groups, namely: group 1, in which there were 1,852 ears which

received myringotomy before the tympanic membrane ruptured; group 2, in which there were 415 ears which received myringotomy after the eardrum had ruptured; and group 3, in which there were 1,843 ears in which the eardrum ruptured and did not receive myringotomy at all.

In group 1, there were 194 mastoidectomies, or 10.4 percent. In group 2, there were 27 mastoidectomies, or 6.5 percent. In group 3, there were 236 mastoidectomies, or 12.8 percent.

In groups 1 and 2, where myringotomy was performed, there were 1,792 ears in which the tympanic membrane was incised once, 339 in which it was incised twice, 101 in which it was incised three times, and 34 in which it was incised four times.

The writer would like to state here that as a general rule he does not favor repeated incisions of the eardrums, as he feels that the bulging of the tympanic membrane, which invites repeated incision, usually is due to the enormous swelling and edema of the mucosa of the middle ear and not due to localized pus, and repeated incision is often not only valueless but that it inflicts useless trauma on the tympanic membrane and middle ear structures.

TABLE I

<i>Ears</i>	<i>Number of incisions</i>				<i>Mastoidectomies</i>		
	<i>1</i>	<i>2</i>	<i>3</i>	<i>4</i>	<i>Unil.</i>	<i>Bil.</i>	<i>Deaths</i>
Incised, 1,852 .....	...	...	...	..	102	45	16
Ruptured and incised, 415...	1,792	339	101	34	25	1	5
Ruptured, 1,843 .....	...	...	...	..	144	46	14
Number of ears .....	4,100						
Average number of days from operation to recovery.....	47						
Number of mastoids averaged.....	313						

About 90 percent of otitic patients in scarlet fever are under eleven years of age. Table II gives the incidence for various age groups for those 2,898 otitic patients. In the group between one and five years inclusive were 1,774, or 61.2 percent; in the group between six and ten years inclusive were 892, or 30.7 percent; in the group between eleven and fifteen years inclusive were 135, or 4.7 percent; in the group between sixteen and twenty years inclusive were 51, or 1.8 percent; and in the group twenty-one years or older were 46, or 1.6 percent.

It has been stated by Borden,<sup>6</sup> with whom the writer concurs, that "it may truly be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of acute symptoms to the last day of convalescence." This is an

TABLE II

<i>Age</i>	<i>Number of patients</i>	<i>Percentage</i>
1-5 .....	1,744	61.2
6-10 .....	892	30.7
11-15 .....	135	4.7
16-20 .....	51	1.8
21 .....	46	1.6
Total number of patients.....		2,868

interesting and important practical characteristic of scarlet fever. Table III shows the week in morbidity of the onset for the group of ears. The first week had 1,021 ears; the second, 990; the third, 598; the fourth, 433; the fifth, 352; and the sixth and thereafter had 716.

TABLE III

<i>Week of Onset</i>	<i>Ears</i>
1 .....	1,021
2 .....	990
3 .....	598
4 .....	433
5 .....	352
6 .....	716
Number of ears.....	4,110
Number of patients.....	2,868

The data here presented correspond closely with those found by other writers. For example, Harries and Gillespy<sup>7</sup> report a series of 290 patients with scarlatinal otitis in which 40.3 percent were under five years of age; 54.5 percent were between five and ten; 8.3 percent, between ten and fifteen; 2.4 percent, between fifteen and twenty; and 3.4 percent, over twenty.

The duration of the period of otorrhea is often annoying to both the physician and the patient. The average number of days of discharge was thirty-six for a series of 1,915 consecutive ears that became dry in the hospital without mastoidectomy. This figure becomes more interesting when one considers that one usually has to decide by the fourth week whether or not a mastoidectomy is indicated.

Gardiner<sup>8</sup> reports that at the Edinburgh City Hospital, where no patient with scarlet fever otorrhea was allowed to leave the hospital, the average stay in the hospital of such patient was 68 days in 1919; 62 in 1920; and 52 in 1921.



In this connection it seems apropos to say something about the rôle of the pharyngeal and faucial tonsils in the development of otitis media in these patients. The nose and throat are undoubtedly the foci of infection in scarlet fever, and the presence of tonsils and adenoids contributes to the severity of the infection. If the infection reaches the middle ear, as is generally supposed, by way of the eustachian tube from the throat, then it is logical to expect that if the infection here could promptly and effectively be eradicated there would be no complicating suppurative otitis media. Unfortunately there is no method of so eradicating the infection. It was expected of scarlet fever antitoxin that it would provide to a reasonable degree such a method, but it is safe to say now that in its present efficacy it does not. On the other hand, infection may, at least in some cases, reach the middle ear by way of the blood stream. In such case it seems that an effective therapeutic method for controlling the nasopharyngeal and anginal infection would still circumvent the development of suppurative otitis media.

Among the 2,898 patients here under discussion there were 169, or 5.8 percent, who had the tonsils and adenoids removed before the development of scarlet fever. In a series of 14,733 consecutive patients with scarlet fever the tonsils and adenoids were absent in 861 patients, or 6.8 percent.

Science has developed no great improvement in the therapeutics of acute suppurative otitis media. The useful principles of treatment which are available today are essentially those available a quarter of a century ago. They may be outlined as follows:

(a) Prevention, by keeping the nose and pharynx free from excessive secretions.

(b) Frequent inspection of the ears, with early myringotomy in every case of bulging tympanic membrane before it ruptures.

(c) Frequent cleansing of the external auditory canal to facilitate drainage.

(d) Consideration of the removal of the adenoids when the ear has discharged for three weeks.

(e) Performing of a simple mastoidectomy in those cases where there is persistent and profuse otorrhea, sagging of the posterior superior canal wall, nipple-shaped perforation, mastoid tenderness and edema, with fever and positive x-ray evidence of surgical mastoiditis.

#### ORBITAL COMPLICATIONS

From the standpoint of the otolaryngologist, there is one outstanding important orbital complication in scarlet fever, namely, orbital cellulitis. Orbital cellulitis is most frequently due to disease

of the paranasal sinuses, and this is the case in scarlet fever, with rare exceptions, such as peridental abscess or cellulitis of the face or forehead. In children, who constitute approximately 90 percent of the scarlet fever census, it is most often due to ethmoiditis. In adults, the frontal sinus is most frequently the seat of the infection.

There are three stages to orbital cellulitis:<sup>9</sup> First, inflammatory edema with or without discoloration of the lids, usually most marked in the upper lid. Second, edema of the lids with exophthalmos, and with or without limitation of orbital movement. Third, the same as the second but with evidence of abscess formation.

In a series of fifty cases of orbital cellulitis, forty-five of the patients were children and five were adults. In all of them except two the orbital swelling occurred in the course of scarlet fever. In seventeen the cellulitis started in the first week of morbidity, fourteen started in the second, ten started in the third, one started in each of the fourth, fifth and sixth weeks. Three started in the seventh, two started in the eighth and one started in the fifteenth weeks.

In all cases except four the orbital swelling was secondary to ethmoiditis. Of the exceptions, frontal sinusitis was the focus in two, both of whom were adults; maxillary sinusitis in one, an adult, and cellulitis of the cheek in one, a child.

In the series of fifty cases of orbital swelling, thirty-five were due to unilateral cellulitis, twenty of the right orbit and fifteen of the left; seven were due to bilateral cellulitis; six were due to unilateral abscess, four of the right orbit and two of the left; and two were due to cellulitis of the right side and abscess (in the same patient) on the left side.

TABLE IV

<i>Orbital cellulitis</i> <i>Unil.</i>		<i>Orbital cellulitis</i> <i>Bil.</i>	<i>Orbital abscess</i>		<i>Orb. cell. &amp; abs.</i>	
<i>Right</i>	<i>Left</i>		<i>Right</i>	<i>Left</i>	<i>Right</i>	<i>Left</i>
20	15	7	11	2	2	

The modern and proper method of treating infections of the orbit has been thoroughly outlined by Porter<sup>9</sup> and Davis.<sup>10</sup> The writer's experience has been mostly with children and the treatment as applied to them in particular may be stated briefly.

The treatment depends on the stage of the disease. In the first and second stages it is conservative. X-ray of the sinuses is desirable. Ice-cold compresses are applied to the affected eye, and the nose is packed with pledgets of cotton wet with 1 percent cocain hydrochloride or 1 percent ephedrin sulphate, two or three times daily. The nasal fossæ must be evacuated frequently with normal saline

solution or by aspirating the secretions with a mechanical suction machine and small rubber catheter, about 10 or 12 French.

When there is evidence of abscess formation or where the edema, exophthalmos and limitation of motion are marked, more radical measures are indicated.

In the milder cases removal of the anterior tip of the middle turbinate and opening of the ethmoid cells, together with an intranasal antrotomy, where there are symptoms of antral disease, will usually be sufficient. In the more severe cases ethmoidectomy by the combined external and intranasal approach is the method of choice.

The writer would like to emphasize that, according to his experience with children, the conservative method, such as outlined for the first and second stage, can nearly always be followed until the condition subsides or abscess formation occurs.

It is proper to stress the fact that an essential step in any operative procedure is to drain the sinus from which the disease spreads to the orbit, and to caution against the inadequacy and danger of making stab incisions into the orbital tissues in order to locate a collection of pus.

#### SUMMARY

Statistics concerning 2,898 scarlet fever patients with acute suppurative otitis media and forty-eight scarlet fever patients with orbital cellulitis are presented.

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## CONSIDERATIONS OF PETROUS PYRAMID SUPPURATION\*

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"The petrous bone now occupies somewhat the position which the mastoid occupied twenty or thirty years ago." This statement was made by Ballance<sup>3</sup> in 1919 and is even more applicable today, for today the subject of petrous pyramid suppuration occupies the center of interest in otology. Our clinical experiences, our anatomical and surgical research, and a comprehensive review of the literature, prompt us to present this paper, which deals with some of the less understood phases of petrous pyramid suppuration.

Infection of the petrous portion of the temporal bone and a spread of the disease to the surrounding structures has been recorded many times in the past two hundred years. In 1740 Morgagni performed an autopsy upon a boy of twelve who had suffered what was probably an acute exacerbation of a chronic otitis media. Part of his account follows: "The pus had entered the cranial cavity, and I was able to demonstrate this to all present by an opening on the petrous portion of the temporal bone which some call posterior and others internal and inferior. For at that surface . . . the caries had made an opening nearly circular in shape and about the size of a lentil bean, etc." Adolph Murray of Copenhagen published his anatomical researches upon the mastoid cells in 1791, at which time he spoke of "innumerable small cells of which some are spread throughout the mastoid, but others extend beneath the upper and posterior walls of the petrous."

Infections of this portion of the temporal bone may be classified as acute, subacute and chronic. The truly acute infection exists infrequently as compared with the subacute. When one speaks of petrositis or petrous infection as it has been described by Kopetzky and Almour and as it has been encountered by many others, one has in mind the subacute type of infection. It is this condition which concerns us in this paper. The chronic long-standing cases of petrous involvement present entirely different problems which do not apply

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here. These cases are asymptomatic for very long periods of time and may terminate with a purulent meningitis or a brain abscess. Cases such as those reported by Friedreich,<sup>17</sup> Crosby Leonard,<sup>36</sup> and Bigger<sup>6</sup> are of this class and are instructive in this connection. The infection may also be classified as localized and diffuse. Kopetzky and Almour<sup>31</sup> first called our attention to the fact that a great majority of acute and subacute infections occur in pneumatized bones. This fact is no longer questioned. In a relatively small percentage of cases the structure of the bone is diploic or a mixture of diploic and pneumatic. The term osteitis as applied to disease of the petrous portion of the temporal bone has come to mean infection in a well pneumatized bone while osteomyelitis implies a more widespread lesion in bones of lesser pneumatization.

#### SYMPTOMATOLOGY

Involvement of the petrous apex is evidenced by certain symptoms, usually after a mastoidectomy has been performed. But this is not always so, for mastoid disease does not always precede or accompany petrous involvement. This occurred in our cases No. 6 and No. 7 and in the case of Uffenorde.<sup>58</sup> There are many variations from the usual clinical picture which will be dealt with later.

The cranial nerves which are most frequently involved are the fifth, sixth and seventh. Occasionally the ninth, tenth and eleventh may be involved when the infection breaks through posteroinferiorly in relation to these nerve trunks.

Perhaps the most frequent and most important symptom is pain which is caused by the involvement of one or more branches of the fifth nerve, most frequently the ophthalmic branch. Pain is encountered in or about the eye and is usually the first symptom. It may occur during the day but is nocturnal as a rule. It may occur only in the early morning hours or may be more or less constant throughout the night. Pain has been noted in the forehead, the upper or lower jaw, and in the temporal region of the affected side. Eagleton<sup>13a</sup> explains the irritation of the ophthalmic branch as due to tension upon the dura surrounding this nerve as a result of the edema and swelling of the vessels of the dura from the nearby disease. We have sought for an explanation of this pain and find it difficult to accept it as being due to direct irritation or to the mechanism suggested by Eagleton. The ophthalmic division of the fifth nerve is situated higher and is more mesial than its other branches. We believe that involvement of the recurrent tentorial branch of the ophthalmic described by Luschka,<sup>40</sup> may be the explanation of this phenomenon. This branch leaves the ophthalmic shortly after it

passes through the superior orbital fissure and traverses the anterior surface of the petrous bone near its apex after which it is lost in the tentorium cerebelli where it terminates. It was interesting that Krause<sup>34</sup> was of this opinion in 1904 when he discussed this symptom. In discussing the frontal headache of the patient whom he successfully operated upon, Krause<sup>34</sup> stated that the superior border of the pyramid and the tentorium were innervated by the ramus recurrens of Arnold (he was probably in error as Arnold's nerve is the auricular branch of the vagus), a branch of the first division of the fifth nerve. He believed that the pain was transmitted from the irritated recurrent branch by way of the ophthalmic branch to its other terminations. He considered that this was borne out by the fact that following his operation when he released the pus from the petrous area, the frontal headache disappeared and did not return. In some cases pain has continued despite operation but this is very likely due to the fact that some infection still remained. In reviewing the case reports one is impressed with the fact that the pain usually disappears within twenty-four hours after operation wherever a cure has been accomplished. When, however, there still remains some disease of the petrous apex and the clinical course does not indicate immediate and uninterrupted recovery, the pain continues until such time as a change occurs in the clinical course which governs a cure.

Brouardel<sup>10</sup> recognized the presence of facial neuralgia in petrous infections as early as 1866 and mentioned the increase in the intensity of the pain when the discharge lessened. Two years later Von Troeltsch<sup>59</sup> called attention to the facial pain which accompanied ear infections. Berger<sup>5</sup> suspected that a caries of the petrous bone existed because of the neuralgia with which his patient suffered. Some years later Ostmann<sup>47</sup> wrote of the pain in and about the eye which has been more recently emphasized by Kopetzky. Increase in the intensity of pain frequently indicates an increase in intraapical pressure and should be properly evaluated.

Since the first paper of Gradenigo<sup>22</sup> sixth nerve involvement has been well understood. Paralysis of the abducens is by no means a constant finding in petrous infections but when it does occur in conjunction with other symptoms and findings its presence is of significance. Now, however, we well know that not every case with a sixth nerve paralysis requires operation.

Seventh nerve paralysis is seen as the first warning of a petrous involvement, perhaps more frequently than is thought. When one considers the close proximity of the facial nerve to the cells which are more or less constantly present in the region of the ampullary end of the superior semicircular canal one can appreciate the fact that a

suppurative process which is travelling along this cell channel to the apex can readily involve the seventh nerve as it begins its course inward. We speak of this area as the superior canal facial angle area. Of significance is the fact that the paralysis is transient as a rule because as the tension of the acute inflammatory process is relieved by the softening and liquefaction which follows, the involvement of the sheath of the nerve resolves while the infectious process is progressing inward. In our anatomic studies<sup>44</sup> we found that the greatest number of pneumatized cell channels originated in this region, while in a review of the literature from which we were able to collect fifty-three well-defined fistulas fourteen began in this location, by far the largest number found in any single location. (See Table 1, showing sites of origin of fistulæ.) A recent experience indicates that the facial nerve can be affected by a diseased tract of cells which originate in the arch of the superior semicircular canal.

TABLE 1.—SITE OF ORIGIN OF FISTULÆ  
Collected cases—total 53

1. Anterior to superior canal .....	14
2. Posterior to superior canal .....	8
3. Posterosuperior border .....	3
	— 25
4. Tubal .....	5
5. Peritubal:	
(a) Superior .....	5
(b) Inferior .....	3
(c) Not mentioned .....	5
	— 18
6. Posteroinferior (jugular) .....	5
7. Arch of superior canal .....	4
8. Posterior—behind posterior canal .....	1
	— 10

This involvement takes place in that portion of the facial nerve which extends from the geniculate ganglion to the internal auditory meatus. Broeckart,<sup>9</sup> Kopetzky,<sup>33</sup> Profant,<sup>49</sup> Friesner and Druss<sup>18</sup> and others have reported cases in which the seventh nerve was temporarily affected. We have had three such experiences.

*Discharge* from the middle ear or mastoid may be scant or absent, or profuse. It will be profuse as a rule but this is by no means constant. The discharge from the postauricular wound or canal is profuse when a fistulous tract or a diseased area of bone is emptying its exudate into the middle ear or mastoid cavity. A fistula may be blocked by granulations even after it has been patent and discharging profusely. In such a case the profuse discharge is no longer present and there is very little if any exudate coming from the wound or the

ear canal. The discharge may be scant or absent when there is very little or no outlet from the petrous to permit the escape of the products of infection. When such is the case the pain is increased. Such a change in the symptom indicates that the exudate is trapped. When the discharge is scant or absent and the pain is not increased but is rather less or absent, it indicates one of two things—either the process is resolving or the infection has eroded through the cortex of the petrous apex. There are variations from the pictures just outlined. The mastoid may be only slightly involved, and the petrous markedly so. The middle ear may be dry and the patient may have fever and a severe headache.

*Temperature* is such as may be expected from an infection which is confined within a small bony space and from which absorption is not rapid. As has been stated before the infection is usually subacute. The temperature ranges between a little above 99 degrees to a little above 101 degrees; in children it may reach 102 degrees. When, however, a complication threatens, the rise of temperature may be the first indication of what is to follow. We have been impressed with the fact that the temperature usually fails to come back to normal in the course of an otitis media or following a mastoidectomy in the cases which are progressing to a petrous infection. This low grade temperature may continue for weeks before involvement of the petrous tip makes itself known. The pulse does not bear a constant relation to the temperature, at times being elevated out of proportion to it. This is more pronounced in cases of longer duration and is no doubt the result of a low grade toxemia. The period of quiescence which Kopetzky and Almour<sup>31</sup> have stressed is of real importance in that it warns the otologist that not only may his patient be far from recovery but also that an intracranial complication may make its appearance at any time. The period of quiescence may be short or long and may at times be absent. It is important to stress the fact that one must be aware that a latent period exists, when after an otitis media of several weeks' duration or after a mastoidectomy had been done some weeks before, the patient maintains a temperature above normal and has a profuse discharge from the ear canal or postauricular wound. A postauricular wound that has not healed after a reasonable period of time has elapsed should make one suspicious. A sustained normal temperature and a profuse discharge with diminishing or absent pain constitute a favorable sign. As an unfavorable sign we would note the combination of persistence of temperature and the persistence or increase of pain with profuse discharge. These with no discharge would indicate a more seriously unfavorable course.



*Labyrinthine irrigation* occurs in some cases: The symptoms of vertigo and nystagmus are due to an inflammation of the cellular structure about the labyrinth causing a perilabyrinthitis. Functional tests of the labyrinth bring normal responses, proving that a true endolabyrinthitis does not exist. When, however, labyrinthine symptoms occur in the presence of a seventh nerve involvement the possibility of serious trouble must be kept in mind. In such a case the seventh nerve may become involved just above the oval window and the labyrinth may be infected by way of this structure.

*Laboratory Data.*—The blood count keeps pace with the temperature range. The leucocytosis is of mild degree, the count being usually between nine and twelve or thirteen thousand. When, however, the temperature rises because of a beginning intracranial complication the white blood cells rise with it, going to the levels which are seen in an irritative meningitis or later a true meningitis.

The same bacteria are found in petrous disease as are found in middle ear and mastoid infections and the same fear and respect is accorded the type three pneumococcus as it has received heretofore. Although this organism is responsible for many deaths due to complications of suppurative otitis media it must not be considered as the causative factor in all fatal cases of petrous apex involvement. Wilkinson,<sup>62</sup> Rollin,<sup>52a</sup> Graham,<sup>21</sup> Grunert,<sup>24</sup> Fowler<sup>15</sup> and others have reported petrous infections by this organism.

The spinal fluid shows no significant findings until a pachymeningitis exists. The eye-grounds are usually normal except where a complicating sinus thrombosis exists. The spinal fluid should be examined in cases which evidence increasing pain in the presence of a diminishing discharge.

The roentgen study is of great importance and is considered the most important single guide other than the combined symptoms. This study frequently furnishes a knowledge of the cellular structure of the petrous pyramid which is essential to an understanding of its disease. Therefore x-ray study in the Stenver and Taylor positions should be carried out to inform us not only of the condition of the apex tip area but also of the cellular channels leading from the middle ear and mastoid cavities toward the apex tip.

*Fistula.*—The entire subject of petrous pyramid suppuration centers about the matter of fistula formation from the middle ear or mastoid areas into the apex. It is obvious that pneumatized bones are best adapted for fistula formation and that diploic bones can contain fistulae also. Our experience has demonstrated that the perilabyrinthine cells are frequently of a diploic type while the apical cells are at the same time pneumatic. The cellular anatomy of the petrous

pyramid has been studied by the authors and by several before them. The most recent anatomical work, which deserves mention, is that of Hagens.<sup>26</sup> He studied twenty-five bones in gross section and twenty-five bones histologically. Seven or 28 percent of the gross specimens had pneumatized apices while 10 or 40 percent of the histological specimens had pneumatized apices. Four of the seven and nine of the ten bones with pneumatized apices presented peritubal cells. The present authors failed to record peritubal cells as such, in their



FIG. 1. A semidiagrammatic reproduction of the sites of origin: (a) anterior to the superior semicircular canal; (b) posterior to the superior semicircular canal; (c) tubal and peritubal region; (d) postero-inferior (jugular) area; (e) arch of the superior semicircular canal, and (f) posterior to the posterior canal.

study, because that study dealt with cellular channels from the periphery to the apex and consequently did not include peritubal cells. Their observation of peritubal cells was carried out only in the pneumatized petrous bones. In the light of our present knowledge it is essential to know not only the well-formed cell channels but also the location of such cells which might be the starting point of a diseased tract toward the apex. Such cells would include the peritubal cells.

The fact that there are certain well-recognized cell channels or groups of cells which favor fistula formation is well established. We have been able to collect from the literature a series of fifty-three fistulae found at operation or at autopsy. They are represented in the accompanying tables (see Figure 1 and Tables 1 and 2.) Twenty-five were in relation to the anterior surface, eighteen were found to originate in or about the eustachian tube orifice, five originated in

the mastoid area posteroinferiorly, four in the arch of the superior semicircular canal and one directly behind the posterior canal. Although none have been recorded, it is possible to have a fistula originate directly above the arch of the superior canal. In such a case the cortex above would no doubt be involved because of the very small space between the arch and the cortex even where cellular structure exists in this location.

TABLE 2.—TABULATION OF FISTULÆ ACCORDING TO AUTHORS

<i>Anterior to superior canal</i>		<i>Peritubal Inferior</i>	
Aloin .....	2		
Bowers .....	1	Graham .....	1
Eagleton .....	1	Mayer .....	1
Lange .....	2	Wilkinson .....	1
Lillie-Williams .....	1		—
Jungert .....	1	Total .....	3
Muck .....	1		
Seydell .....	1		
Uffenorde .....	2		
Authors .....	2		
	—		
Total .....	14		
<i>Posterior to superior canal</i>		<i>Peritubal Not mentioned</i>	
Graham .....	1	Boemer .....	1
Knapp .....	1	Friesner-Druss .....	1
Kopetzky-Almour .....	3	Kopetzky-Almour .....	1
Lange .....	1	Leutert .....	1
Lillie-Williams .....	1	Ramadier .....	1
Lombard .....	1		—
	—		
Total .....	8	Total .....	5
<i>Tubal</i>		<i>Arch of superior canal</i>	
Barr .....	2		
Fowler .....	1	Freneckner .....	2
Grunert .....	1	Weeth .....	1
Kopetzky-Almour .....	1	Authors .....	1
	—		—
Total .....	5	Total .....	4
<i>Peritubal Superior</i>		<i>Posteroinferior (Jugular)</i>	
Grunert .....	1	Bowers .....	1
Kopetzky-Almour .....	2	Eagleton .....	1
Uffenorde .....	1	Hilgerman .....	1
Wilkinson .....	1	Muck .....	1
	—	Sunde .....	1
Total .....	5	Total .....	5
		<i>Posterior Behind posterior canal</i>	
		Kopetzky-Almour .....	1
		<i>Posterosuperior border</i>	
		Friesner-Drus .....	3

Fistulæ into the petrous bone were encountered as early as 1847 when Fletcher<sup>14</sup> reported the following experience:

A strong boy of thirteen developed an acute otitis media on September 1. On the seventh day an abscess was opened over the mastoid area from which a fluid discharged freely until the day of his death. On the eleventh day symptoms developed with fever and delirium. He became semicomatose and died on the twenty-sixth day. The bone presented four openings each of which directly or indirectly communicated with the middle ear.

Haberman<sup>25</sup> reported what is probably the earliest finding of a fistula at operation (1897). In performing a third and radical operation upon a six-year-old boy with petrous symptoms he was able to penetrate between the semicircular canals to a spot opposite the internal auditory meatus where he came in contact with another necrotic focus and also at the same time struck an arterial vessel which prevented further interference. The patient recovered. In 1900 Muck<sup>43</sup> encountered two fistulæ while operating upon a man of forty-eight. These he enlarged but despite this the patient did not recover until the suppurative focus worked its way into the retro-pharynx where it was recognized and evacuated.

That we should expect to find fistulæ in disease of bone is a pronouncement, the basis of which can be found in every textbook of pathology. The pneumatized cell channels would favor fistula formation more readily than any other type of bone structure. The disease extends to the apex tip area by means of a process of coalescence along a preexisting channel of cells. Fistulæ may end blindly before they reach the apex area. Or a fistula originating in the tubal or peritubal area may extend into the carotid canal where the disease progresses to penetrate the canal and reach the apex. In cases where the fistula ends blindly before reaching the apex it may empty itself spontaneously or it may break through in a direction other than that of the axis of the original tract. It is likely that this type of infection is responsible for the perforations of the anterior (superior) and posterior surfaces peripheral to the apex (see Figure 1.) This visualizes the various points of origin of the fistulæ. In addition we have tabulated the various fistulæ according to location and author (see Tables I and II.) We strove to include only such cases in which the fistula was clearly defined and readily understood by us. It is possible that our interpretation of the location of a given fistula may not coincide with that intended by the authors. Careful deliberation was given each fistula and the location mentioned in the tabulation is according to our best interpretation. Unfortunately many descriptions were incomplete and some were confusing. The fistula described by O. Mayer<sup>41</sup> which has been called a hypotympanic



fistula by some has been included under the heading of peritubal inferiorly placed fistula.

An analysis of fifty-three fistulæ discloses that twenty-nine were in relation to the superior semicircular canal; three of these were reported as occurring in the posterior superior border which is in reality an extension backward of the cells directly behind the superior canal. Four of the twenty-nine fistulæ were found in the arch of the canal. There were eighteen fistulæ in and around the eustachian tube mouth. Five were found just below the posterior canal and are otherwise classified as jugular bulb fistulæ. Of interest is the relatively large number of multiple fistulæ which have been recorded, six up to the present time. Table 3 shows the distribution of these multiple fistulæ.

TABLE 3.—MULTIPLE FISTULÆ; LOCATIONS AND AUTHORS

Barr <sup>4</sup> :	Two fistulæ originating in mouth of the Eustachian tube
Bowers <sup>8</sup> :	Anterior to superior canal, posteroinferior
Grunert <sup>24</sup> :	Peritubal, superior; tubal
Kopetzky-Almour <sup>30</sup> :	Posterior to superior canal, peritubal
Muck <sup>43</sup> :	Anterior to the superior canal, posteroinferior; retropharyngeal abscess
Wilkinson <sup>62</sup> :	Peritubal, inferior; peritubal, superior

A great variety of fistulæ are represented in the accumulated experiences of the authors mentioned in the tabulation above. This suggests that there is very likely a fistula or tract of softened cellular bone in every petrous infection which causes the symptoms which are now well known. The significance of this in formulating a plan for the surgical management of this condition is at once apparent. It is also obvious that no one surgical procedure can be applied to every case. The significance of the presence of fistulæ prompts the rule: "*Seek the fistula.*"

A review of the cases reported indicates the route along which the infection travelled and the locations where a break occurred through the cortex of the petrous bone. Most of the erosions occurred through the anterior (superior) surface of the apex. Friedrich reported such a case in 1829. Similar experiences have been reported by Goris,<sup>20</sup> Knapp,<sup>30</sup> Jouty,<sup>28</sup> Eagleton,<sup>13</sup> Kopetzky and Almour,<sup>33</sup> and others. These erosions are frequently associated with meningitis or brain abscess. Eagleton<sup>13</sup> had two cases where erosions occurred through both the anterior and posterior surfaces of the apex. Morgagni, in 1741, Graham, Fremel and others, reported posterior perforations of the cortex of the petrous apex.

When the inferior aspect is eroded a retropharyngeal abscess occurs. We have collected nine such instances (see Table 4.)

TABLE 4.—RETROPHARYNGEAL ABSCESSSES

According to Authors

Aloin .....	1
Bowers .....	1
Eagleton .....	2
Greenfield .....	1
Grunert .....	1
Muck .....	1
Seydell .....	1
Authors .....	1
<hr/>	
Total .....	9

It would appear that the formation of a retropharyngeal abscess in petrous apex empyema is indicative of a favorable outcome if one is to judge from the case reports available. The relatively greater frequency of erosions through the anterior surface of the petrous apex is responsible for the attempts which have been made to cure this condition by elevating the temporosphenoidal lobe of the brain down to the petrous tip and placing a drain in this area with the hope that a break will occur here and that the pus will drain off without the meninges becoming involved. Streit<sup>55</sup> attempted to do this in 1902 while Broeckart<sup>9</sup> and Jouty<sup>28</sup> were successful some years later. In view of what we have learned in recent years this procedure is not to be recommended unless the additional step of opening the apex tip is contemplated.

The complications of suppuration of the petrous pyramid are too well known to justify lengthy consideration here. Meningitis and abscess of the brain are well known as complications while involvement of the venous channels occurs with much less frequency. That the cavernous sinus is occasionally involved can be seen from the reports of Knapp,<sup>30</sup> Hilgerman,<sup>27</sup> Eagleton,<sup>13a</sup> Brunner,<sup>11</sup> and Courville and Nielsen.<sup>12</sup> Atkins<sup>2</sup> reported a case in which there was a thrombosis of the inferior petrosal sinus. When sepsis supervenes upon the clinical picture of petrous infection involvement of these venous blood channels must be thought of. The carotid venous plexus and at times the carotid artery itself may become the site of a thrombotic lesion where the infection has invaded the carotid canal.

A great deal of confusion exists concerning the indications for operative drainage of the petrous apex. As yet a definite plan of

management based upon the diagnostic data available has not been formulated. The following is an attempt to tabulate a plan of management according to indications.

PLAN OF MANAGEMENT ACCORDING TO INDICATIONS

	<i>Conservative</i>	<i>Operative</i>	<i>Urgently operative</i>
Pain	Not increasing	Nocturnal increasing	Increasing
Temperature	Little or none	Static or increasing	Static or increasing
Discharge	Profuse or scant	Absent or diminished, m. b. profuse	Increased or diminished, may cease
X-ray	Negative or suggestive	Positive or suggestive	Positive or suggestive
Spinal fluid	.....	Negative	Signs of meningismus

As has been stated before, pain in and about the eye or in the forehead region which occurs at night and recurs, is indicative of disease of the petrous apex and should not be disregarded. A diminution of discharge together with an exacerbation of pain constitutes a pressing indication for surgery. If the pain is indefinite or cannot be well understood because of a lack of cooperation or a feeling that there is a lack of cooperation one should study carefully the other signs and symptoms. If the temperature should rise, if the discharge which was absent or scant becomes profuse and continues so or becomes less again, if the patient looks ill, an x-ray of the petrous should be made and disease should be seriously suspected. Pain is the outstanding symptom. X-ray study will usually assist in making a diagnosis for there is usually a definite shadow upon the film when empyema of the apex exists. Occasionally the evidence is so striking that it constitutes a most positive indication for surgery. In case 8 (see figures 5 and 6) no one can doubt that the entire apex area has been destroyed and is absent in the film. The basis for operative surgery then would be first, pain, nocturnal and recurring, together with x-ray evidence, and the other symptoms which have been mentioned. Immediate operation is indicated when in addition to the above there is an increased spinal fluid pressure and an increased cell content of the spinal fluid, with little or some clinical

evidence of meningeal irritation. A mastoid wound which continues to discharge for a month or more after operation should draw attention to the possibility of petrous disease.

#### TREATMENT

We believe that many papers which have been appearing with more or less regularity justify the statement that a policy of watchful waiting is extremely dangerous in many cases. The indications for operation upon the petrous pyramid would consist of those signs and symptoms which indicate a trapped collection of pus in the apex area. These have just been considered. In this connection the statement of Ballance<sup>3</sup> is especially applicable. He stated that "the danger lies not so much in operation as in delaying operation." Many otologists of distinction have warned of the dangers of infection of the petrous pyramid and apex and of the necessity of dealing boldly with such infections. In 1868 Von Troeltsch<sup>59</sup> said: "I know of scarcely a part in the human organism which in all directions is so surrounded by important parts and organs, and in which accumulations of pus should be so avoided, as the middle ear."

The clinical experiences, the postmortem observations and the histological studies of a large number of observers assist us to better understand the manner in which the infection progresses. With the advantage of this knowledge we are better able to plan the treatment of infection of the petrous bone with a trapped empyema of its apex.

Richards<sup>52</sup> was in advance of his time when in 1918 he reported his results in operating upon eight petrous pyramids. He carried out a complete removal of the bone in five cases of cancer and three of suppurative disease. His paper is of value to those interested in this subject and his procedure should be given consideration when one is confronted with a diffuse osteomyelitis of the petrous.

Of specific methods of surgical attack there are at present available the following: (1) The technic of Eagleton<sup>13</sup> in which the surgeon attempts to attack a spreading infection by extensive removal of bone, by the exposure of the petrous pyramid anteriorly and posteriorly in addition to exposure of the apex and the opening of its anterior cortical surface by means of a hook. This operation requires a radical mastoidectomy. (2) Kopetzky and Almour have had a large series of successes with their operation. After performing a radical mastoidectomy they reach the petrous apex by creating an artificial fistula through the triangular space between the cochlea and the carotid canal. (3) Freuckner<sup>16</sup> proposed reaching the apex through the arch of the superior canal while (4) Voss<sup>60</sup> has advo-



cated a radical mastoidectomy with the additional removal of bone to expose the epitympanic region. (5) Ramadier<sup>50</sup> and his associates have performed what appears to be an extremely radical and complicated operation upon the cadaver. After a radical mastoidectomy the tegmen of the middle ear and antrum are removed; in addition part of the glenoid tubercle is resected so that the petritubal area is well exposed. Some bone in this region is now removed to expose the carotid canal. The artery is removed from its bed and the apex is attacked through the canal. The present authors have proposed a technic<sup>45</sup> which enters the anterior wall of the petrous apex with a special gouge. The exposure and operative technic are made easier by the presence of certain landmarks on the posterior border of the petrous. This operation is done only after a thorough simple mastoidectomy has been performed and aims at the preservation of the patient's hearing at the same time as evacuation of the apex area is accomplished.

One need only point to the experience of Muck,<sup>43</sup> who despite the fact that he encountered two fistulæ and enlarged them, did not obtain a cure until a retropharyngeal abscess formed which he incised and evacuated, or to our own experience in which, despite the finding and enlarging of a fistulous tract the patient nevertheless developed signs of meningeal irritation within the next twenty-four hours. In Ruttin's<sup>53</sup> case the dura was elevated down to the apex but no abscess was found. The apex was again approached unsuccessfully through the middle ear by the so-called American operation until a track was finally forced through the bone between the semicircular canals upward and inward through the bone forming the roof of the internal auditory meatus to find a collection of pus. These experiences and others indicate that no one procedure is applicable to all cases. It is therefore important that one should have a definite plan of procedure in mind when he encounters a trapped infection of the apex tip area.

We, therefore, advocate the following plan of procedure. If, as is readily granted, the finding of a fistula and its enlargement so frequently results in a cure we must first try to find such a fistula. Fifty-three fistulæ were collected which occurred in nine different locations. The greatest number were found anterior and posterior to the superior canal. The next greatest number were located in relation to the orifice of the eustachian tube.

We perform a thorough simple mastoidectomy in which the bone in the solid angle between the horizontal and superior semicircular canals is removed exposing as much of the perilabyrinthine area as is

possible. This entails the removal of the tegmen of the mastoid and of the antrum and the removal of the cellular bone in Trautmann's triangle. This brings into view the superior semicircular canal, and most of the horizontal and part of the posterior canal. A systematic search is now made for fistulæ. The arch of the superior canal and the region anterior and posterior to it are investigated, also the space directly above it if such exists. Then the region below the posterior canal and directly behind it are probed. A small-sized Yankauer eustachian curet is of value as a probing instrument. If a fistula is not found by the above technic the following plan is used. The temporosphenoidal lobe is elevated and the anterior surface is exposed down to the tip. The tip is then uncapped by means of special instruments. We prefer to avoid the performance of a radical mastoidectomy especially in children. Those who wish, however, may perform a radical mastoidectomy and search for fistulæ in the tubal and peritubal areas. If a fistula is not discovered after the performance of the radical operation the operator now has the choice of the procedure advocated by the authors, or that of Kopetzky and Almour<sup>32</sup> or Eagleton's<sup>13</sup> operation. Where a profuse discharge exists there is more justification for the radical operation and the search for a fistula in the tubal and peritubal area. If, however, there is no profuse discharge but a scant or even an absent discharge one should at once attack the apex area after the simple mastoidectomy.

When the apex area is entered by means of a previously existing fistula or by one that is artificially made, the otologist should be prepared to expose the apex from above and uncap it if the postoperative course is not satisfactory. This is necessary to make certain that an erosion through the cortex does not exist and with it an epidural abscess.

#### SUMMARY OF OPERATIVE MANAGEMENT

1. Perform as thorough a simple mastoidectomy as is possible.
2. Search for fistulæ: a. Anterior to superior canal. b. Posterior to superior canal. c. In arch of superior canal. d. Above arch of superior canal. e. Below the posterior canal. f. Posterior to the posterior canal.
3. If none are found perform middle fossa exposure and uncap apex. Or, perform a radical mastoidectomy and search for tubal and peritubal fistulæ.
4. If fistula is found and postoperative course is unsatisfactory uncap apex tip after elevating temporosphenoidal lobe.

We have found it advantageous to remove the cortex of the anterior surface down to the apex tip where a fistula is encountered which is in close proximity to the anterior surface cortex.

The following cases are reported:

*Case 1.—Acute otitis media, mastoidectomy (on twenty-second day), petrous operation (ten weeks later), recovery.*

K. N., male, white, age six, began his illness with a right earache and discharge on March 15. Mastoidectomy was performed twenty-two days later. At this time a coalescent mastoiditis was encountered in a well-pneumatized bone. Granulations were found upon the wall of the sigmoid sinus. The boy was discharged on the tenth day with a slight discharge from the postauricular wound and from the ear canal, and a negligible temperature. The foregoing relates to his history before he was admitted to our service on May 27 because of pain over his right forehead and behind the right eye for three days. The pain was much worse at night. A profuse discharge was present coming from both the canal and the wound. His temperature was 102.6 degrees. The blood count and the spinal fluid examination were normal. X-ray study revealed a moderate clouding of the apex area of the petrous. The temperature returned to almost normal at the end of two weeks, while his pain had left him on the third day after admission. He was accordingly sent home on June 10 because he appeared to be making a spontaneous recovery. On June 19, however, he was readmitted because of severe pain behind the right eye and over the right forehead which had persisted during the past three days. A profuse discharge was again present as noted before. The blood count which showed 9,000 white blood cells at the last examination was now 13,600 and the polymorphonuclears had risen from 67 to 76 percent. Spinal fluid findings were again negative. There was now a bilateral papilledema present which was more marked on the right side. X-ray studies in both the Taylor and the Stenver positions disclosed what appeared to be a large cavity filled with pus in the region of the apex. On the next day (June 20) operation was carried out upon the petrous apex under avertin anesthesia. A vertical incision was made from the upper extremity of the postauricular incision directly upward. The skin, muscle and periosteal tissues were retracted. An area of bone about five centimeters in diameter was removed from the squamosa so as to expose the temporosphenoidal lobe of the brain. The dura was now elevated down to the tip. The apex was uncapped and yielded a small amount of pus which was admixed with blood and which gushed into the wound under pressure. A rubber tissue drain was placed in the cavity and allowed to protrude from the external wound. The wound was closed except for the lower end of the mastoid incision. The eye pain disappeared three days later and the papilledema was gone at the end of five days. The patient gradually recovered and was discharged completely well on July 29.

*Summary.*—April 6. Mastoidectomy—after acute otitis media of twenty-two days.

May 27. Readmitted—pain in eye; temperature 102.6, profuse discharge.

June 10. Apparently well—discharged.

June 19. Readmitted—bilateral papilledema. Petrous x-ray positive.

June 20. Petrous operation by middle fossa route.

July 23. Discharged, cured.



## COMMENT

This case demonstrates the conservative management of a case and the necessity for recognizing the significance of severe pain in the head. This symptom together with the papilledema and positive x-ray findings were the indications for operation.

*Case 2.*—This case was reported in a separate paper<sup>40</sup> but is repeated in brief because of certain unusual aspects.

A female, colored girl of twenty-two, was admitted April 10, 1934, complaining of sore throat, headache, pain in the left ear and fever of five days' duration. Sixteen days later a simple mastoidectomy was performed because of profuse discharge, headache, a sagging superior canal wall, and pain and tenderness on pressure over the mastoid area. A coalescent mastoiditis in a well-pneumatized bone was disclosed at operation. On May 7 during a post-

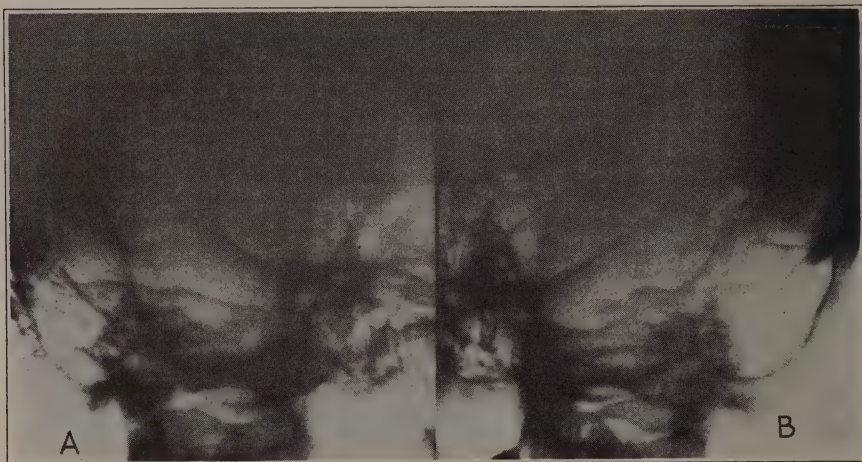


FIG. 2 (*Case 2*). Roentgenogram showing clouding of the area of the apex tip and the perilabyrinthine area of the left side as compared with the normal structures of the other side. Note the loss of definition of the roof of the apex cell.

operative course which had been uneventful the temperature rose to 101 and pain appeared behind the left eye. Six days later the temperature rose to 104.8 degrees while the eye pain persisted. A chill preceded this rise in temperature. A profuse discharge was coming from both the wound and the external canal. X-ray study revealed a loss of outline of the superior border of the petrous apex. On the next day (May 14), eighteen days after mastoidectomy, the wound was explored, and the lateral sinus exposed and found normal in appearance. A black spot indicating necrosis was encountered above the level of the external canal and external to the plane of the superior canal. A probe was inserted in this region and its withdrawal was followed by a gush of pus. The fistula thus uncovered was enlarged and a drain was inserted. On the following day the patient presented signs of meningeal irritation; the neck was rigid, positive Kernig and Babinski signs were present, the spinal fluid was under increased pressure and 325 cells were present in each cubic millimeter, of which 75 percent were polymorphonuclear. A second operation



was performed which consisted of a removal of the diseased bone adjacent to the fistula, and of the cortex above it. The pain disappeared on the next day and the temperature gradually returned to normal on the fifteenth day after operation.

*Summary.*—April 10. Admitted, pain in left ear, fever, headache.

April 26. Simple mastoidectomy.

May 7. Pain behind left eye.

May 13. X-ray of petrous-positive, chill, temperature 104.8.

May 14. Fistula found anterior to superior canal. Gush of pus obtained.

May 15. Signs of meningeal irritation. Erysipelas of postauricular region. Operation, removal of cortex above fistula and removal of adjacent diseased bone. Recovery uneventful.

*Case 3.*—This case and the one which preceded it were recently reported.

A girl of seven years was admitted to the hospital May 12, 1934. The following are the summary and comment for this case:

May 12. Admitted—otitis media for three days.

May 24. Discharged—to outpatient department because there were no signs of mastoiditis.

June 15. Readmitted—pain right temple and a temperature of 103.

June 24. Right external rectus paralysis makes its appearance. Pain over right eye.

June 29. X-ray of petrous—positive. Operation upon petrous contemplated. Preliminary thorough simple mastoidectomy performed as first step which disclosed a fistula anterior to the superior canal. Overlying cortex of anterior surface removed.

*Course.*—Pain disappeared day after operation. Temperature normal on fifth day. Ear dry in two weeks.

#### COMMENT

After seven weeks of a mild otitis media and without signs of a surgical mastoiditis this child developed signs of petrous pyramid suppuration. A petrous operation was indicated by the presence of pain over the right eye, especially at night; the positive x-ray shadow, the long-standing otitis media and the low-grade sepsis. A previous mastoidectomy had not been performed. A fistula was found anterior to the superior canal and in relation to the anterior surface cortex. The overlying cortex and adjacent softened bone were removed. This influenced a more rapid recovery.

*Case 4.*—Simple mastoidectomy, radical mastoidectomy, uncapping of apex, lateral sinus thrombosis, perilabyrinthitis, spontaneous evacuation of retropharyngeal abscess, recovery.

E. O., a colored male, twenty-eight years of age, was referred to us on February 1, 1934. He had had a simple mastoidectomy performed on January 4 because of temperature of 103 degrees, pain in the left ear, and discharge for eight weeks. On January 26 a radical mastoidectomy had been performed because of headache. At this time the operator noted that the bone in the region of posterosuperior border of the petrous was softened.

On February 5 he complained of severe frontal pain. At the same time there was a profuse foul discharge from the wound. The headache gradually disappeared and the patient was discharged twenty-nine days after his radical mastoid operation, on February 24. He was readmitted March 1 with a dry ear but with pain over the left side of his head and over the left eye. His temperature was 102 degrees. He was operated upon the next day. The temporosphenoidal lobe of the brain was elevated, the apex tip was exposed and its roof uncapped with a special gouge. This was accompanied by a gush of pus which was admixed with blood. A rubber drain was inserted into the cavity and permitted to extend from the mouth of the wound.

On the following day there was still pain over the left eye. There was also a horizontal nystagmus to the opposite side. Hearing was present in the left

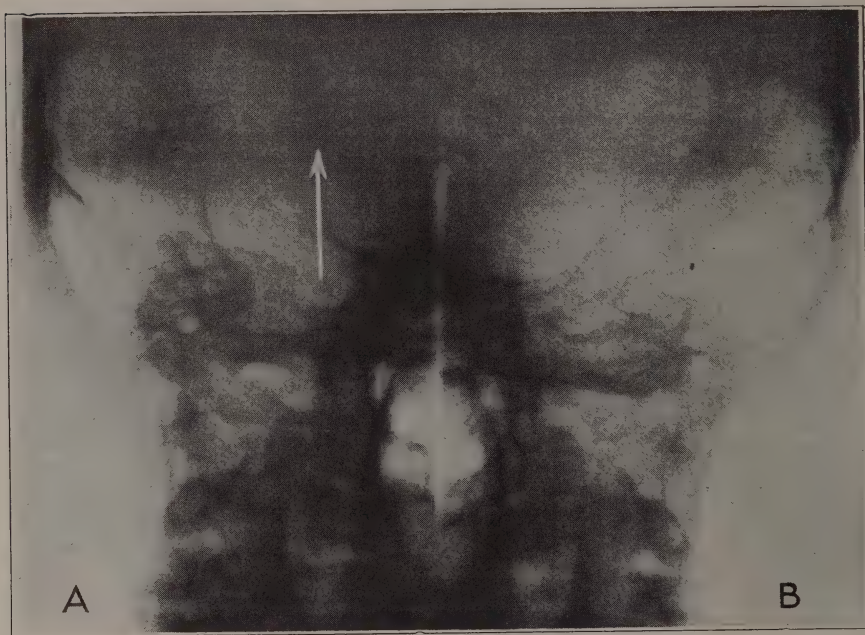


FIG. 3 (Case 4). Roentgenogram showing haziness of the area of the apex tip and the perilabyrinthine area as compared with the same areas on the uninvolvement side.

ear. Two days later there was a slight facial weakness of the same side. The nystagmus disappeared after two days but reappeared a week later. At this time the temperature was 102 and a painful swelling of the left ankle made its appearance. The wound was reopened and the perilabyrinthine area thoroughly scrutinized. Nothing significant was found. The lateral sinus was found apparently normal at this time. Three days later the facial involvement had disappeared. On April 13, fifteen days after the last operation, he had been suffering with severe frontal headache for three days. On that day he had a chill and his temperature reached 104 degrees. He vomited and his neck was slightly rigid. Neurologic study revealed no findings other than a slightly rigid neck. On the same day he complained of severe pain in the left chest

posteriorly; he coughed and expectorated three ounces of blood. This was considered as evidence of a probable infarct of the lung. The spinal fluid was hazy, the globulin was three plus, Benedict's solution showed reduction, and there were 400 cells to each cubic millimeter of fluid of which 75 percent were polymorphonuclear cells. Organisms were not found on smear or culture. (April 13.)

Operation was undertaken upon the lateral sinus. Free bleeding was obtained from the torcular end but not from the jugular end of the sinus. The jugular vein was ligated. Four days later a definite hyperesthesia of all three divisions of the fifth nerve was noted. On April 23 edema of the left posterior tonsillar pillar was noted. This edema extended upward into the nasopharynx along the lateral pharyngeal fold of that side. We now felt that pus trapped in the petrous bone was trying to make its way into the pharynx. Palpation revealed no evidence of a mass. At this time the patient complained of pain in the left side of the throat which was aggravated when swallowing. X-ray study of the nasopharynx was reported as follows: "Above and in front of the first cervical vertebra there is an increased shadow in the upper part of the pharynx encroaching upon the nasopharyngeal space." The nasopharynx and pharynx were watched daily. On the second day after the appearance of the pharyngeal edema and pain the patient expelled a large quantity of pus and blood while blowing his nose. Immediately after this there was a definite change in the appearance and attitude of the sufferer. He had no more complaints and from that time on he made an uneventful recovery.

*Summary.*—January 5. Mastoidectomy for left ear pain, discharge for eight weeks.

January 26. Radical mastoidectomy.

February 1. Referred to authors.

February 5. Severe frontal pain; profuse discharge.

February 24. Discharged improved.

March 1. Readmitted. Ear dry—pain over eye and left side of head.

March 2. Petrous operation—uncapping of apex cell. Small quantity of pus evacuated.

April 13. Eye pain persists intermittently. Chill and rise of temperature to 104. Infarction of lung—hemoptysis. Neck rigid. Spinal fluid—400 cells, 75 percent polymorphonuclears. Operation—thrombosed lateral sinus found.

April 23. Edema of left posterior tonsillar pillar. X-ray positive for early nasopharyngeal abscess.

April 25. Retropharyngeal abscess ruptures while blowing nose. Recovery.

#### COMMENT

This patient had a well-pneumatized mastoid and petrous process and suffered with infection of the petrous pyramid for at least three months. Our operation though well intentioned was of no value in this case nor would any operation have helped toward the patient's recovery. It remained for the development of a retropharyngeal abscess to rescue this patient from a fatal termination by meningitis.



*Case 5.—Mastoiditis, erysipelas, myocarditis, acute osteomyelitis of the petrous pyramid spreading to the body of the sphenoid.*

C. W., a white man, sixty-eight years of age, walked into the hospital July 3, 1933. His illness began six weeks before with throbbing pain in the right ear. There had been no discharge until two weeks before. Examination revealed a moderate amount of mucoid discharge coming from a high and a low perforation. There was a definite loss of hearing while the labyrinth reacted normally. There was no mastoid tenderness and a pneumococcus type three infection was suspected. He was suffering with severe frontal and occipital headache which was more pronounced at night. X-ray study revealed some clouding of the perilabyrinthine and apex areas. A mastoidectomy was performed. At the time of operation granulations were found covering the dura of the posterior fossa and the sinus wall, an epidural and a perisinus abscess being present. For the seven days following the operation the temperature was only slightly elevated. On July 13 the temperature rose to 103 degrees. During the same day the patient was greatly depressed, he complained of pain in the right ear and in the right side of the head. Because of the rise of temperature to above 104 and because of the findings at operation a sinus thrombosis was suspected. Spinal fluid examination at this time was negative. Two days later there was a profuse discharge from the wound, the pain was again present over the frontal and occipital regions of the right side. At the same time erysipelas of the face was diagnosed. Examination by the medical service elicited the fact that a myocarditis was present and the possibility of a septicemia was considered. Blood cultures were negative at this time. He now had a spreading erysipelas which had involved his face and neck and right upper extremity. On July 19, sixteen days after admission, the patient succumbed to the severe strain of his illness.

Autopsy limited to the head revealed an intact dura throughout; intense edema and congestion of the pia was present. In the region of the right mastoid and petrous bone there was fluid pus which flowed freely from a posterior perforation of the apex area. The petrous bone was found markedly necrosed in its perilabyrinthine area and there was marked rarification throughout. The necrosis had involved the apex tip and had extended to the body of the sphenoid. Anatomic diagnosis: erysipelas of face and neck and right upper extremity; leptomeningitis, osteomyelitis of the petrous bone and body of sphenoid.

#### COMMENT

Our lack of experience kept us from operating upon this patient's petrous pyramid. Of interest is the fact that a sixth nerve paralysis did not make its appearance despite the extensive disease which went beyond the petrous tip area.

*Case 6.—Right mastoidectomy; bilateral external rectus paralysis, left mastoidectomy, symptoms of petrous disease, recovery without operation.*

H. K., a white girl of sixteen, was operated upon elsewhere for a right mastoiditis on January 10, 1933. She was admitted to the service two months later complaining of pain in the forehead and behind the right eye which had occurred nightly for the past two weeks. Her white blood cells numbered



7,100 with 63 percent polymorphonuclears present. On April 1 a revision of the mastoid was performed; some infected cells were found in the region of the mastoid tip and the zygoma. The sinus plate was removed at this time because it was found to be soft. The temperature remained between 98 and 100 degrees for the next two weeks. The eye pain continued. On April 15 she complained of double vision; this was found to be caused by a right external rectus paralysis. A bilateral papilledema was now discovered which was more pronounced on the left side. On April 21, the patient developed an acute pharyngitis, the temperature at this time going to 102.4 degrees. At this time also the patient called our attention to her pain in the other (left) eye and on examination a left external rectus was found in addition to the same lesion of the right side. The spinal fluid was negative. The left ear began to ache on the twenty-sixth and discharged spontaneously the same day. The left ear discharged profusely, there was tenderness on pressure, with a sagging superior canal wall and severe pain at night. The right ear discharged but little, the external rectus on this side continued paralyzed. Bilateral papilledema continued. For a few days there had been pain behind both eyes. On May 5 the pain behind the eyes was gone but the bilateral paralysis continued. A left mastoidectomy was performed, the zygoma was cellular and extensively involved. Three days later it was noticed that the left external rectus paralysis was improving. X-ray study which had been made at the time of the recent operation revealed that the left apex of the petrous was decidedly opaque. On June 3 the left external rectus function was normal while that of the right side was improving. On this day she was discharged from the hospital. Two weeks after discharge the right rectus palsy had entirely disappeared.

*Summary.*—January 10. Right mastoidectomy after an otitis media of ten days.

March 23. Admitted to service with pain in eye; temperature 101, profuse discharge.

April 13. Bilateral external rectus paralysis.

April 26. Pain, left eye.

April 28. Left otitis media.

May 4. Pain behind both eyes.

May 5. Left mastoidectomy.

May 8. Left external rectus recovering function. X-ray suggests right petrous tip involvement.

June 3. Discharged recovered; no petrous operation.

#### COMMENT

This was one of our first experiences with petrous infection. Although the patient made a complete recovery after having had pain behind both eyes and paralysis of both sixth nerves it is likely that we would have explored the right petrous apex if we had known more about the subject of petrous infections. The left external rectus involvement came on before the left acute otitis media so that we infer that this paralysis was due to disease of the right petrous tip.

*Case 7.—Acute otitis media, facial paralysis, petrous apex empyema, epidural granuloma, meningitis, death, brain abscess found postmortem.*

A child of nine had a left otitis media of thirty days' duration when she suddenly developed a left facial paralysis. A simple mastoidectomy was performed at which time a pneumatized mastoid process only very slightly involved was found. The operation was carried into the region of the superior semicircular canal. She recovered from the operation, the wound healing uninteruptedly while the facial paralysis disappeared on the seventh day. On the twelfth day after discharge and the twenty-third day after the mastoidectomy a left external rectus paralysis appeared and with it pain in the left frontal region. At this time the wound was healed, the ear was dry, the hearing was apparently normal. The headaches continued for three days when an operation upon the petrous was permitted. On the second day x-ray study revealed evidence of loss of continuity of the roof of the apex tip (see Fig. 4).

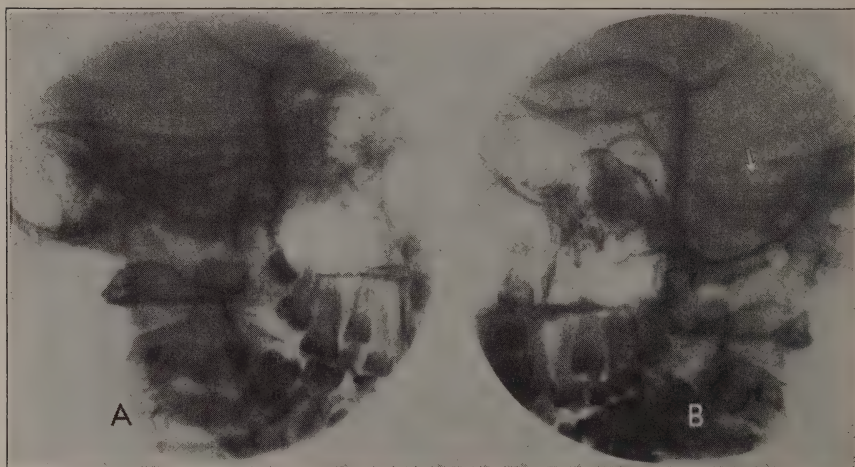


FIG. 4 (Case 7). Roentgenogram taken before operation. The arrow indicates absence of the bony roof of the area of the apex tip. A granuloma was found plugging this area.

*Operation.*—The anterior surface of the apex was exposed and a granuloma was found plugging an erosion through the roof of the apex cell. The removal of this granuloma was followed by a gush of pus. Postoperative course; the headache disappeared immediately after operation. During the next few days there were three small hemorrhages from the wound which were of no consequence. The temperature was irregular and reached as high as 103 degrees during the first week. Albumin was found in the urine daily and vomiting occurred occasionally. Neurologic examinations were repeatedly negative. On the thirteenth day after operation, however, she suddenly developed a headache, neck rigidity and hyperactive reflexes generally. The spinal fluid was removed under pressure and contained 1,800 cells per cubic millimeter, 85 percent of which were polymorphonuclears. The streptococcus hemolyticus was isolated on smear and culture. The patient rapidly succumbed to her infection within twenty-four hours after the onset of her meningitis. Examination (postmortem) through the wound of the middle fossa revealed the pres-

ence of a previously unrecognized temporosphenoidal lobe abscess. At no time were there any signs or symptoms present which might have led us to suspect its presence.

*Summary.*—April 8. Mastoidectomy because of facial paralysis and otitis media of thirty days' duration.

April 19. Discharged with ear dry and wound healing.

April 28. Readmitted; pain in forehead and left external rectus paralysis for two days. Operation—granuloma found plugging roof of apex cell. Pus under pressure released.

May 12. Onset of meningitis; hemolytic streptococcus present.

May 13. Deceased. Investigation of wound revealed unsuspected presence of temporal lobe abscess.

### COMMENT

With a middle ear inflammation which had almost completely subsided and with no evidence of mastoid involvement a facial paralysis developed. This was interpreted as a spread of infection inward toward the petrous apex. A simple mastoidectomy was performed after which the facial paralysis cleared up. Twelve days after operation with a dry middle ear and an almost completely-healed wound signs of a petrous involvement were noted. X-ray study demonstrated a loss of continuity of the superior border of the apex cell area. Operation was carried out on the petrous and a granuloma was found plugging the roof of the apex cell. Removal of the granuloma was followed by a gush of pus. By all the rules which govern the prognosis in such cases we were led to expect a recovery, but on the fourteenth day after the second operation meningitis made its appearance. The patient succumbed within thirty-six hours. At no time were there any neurologic signs pointing to a possible brain abscess which was found on investigation of the wound area after death.

*Case 8.*—Otitis media, simple mastoidectomy, exposure of superior canal area, finding of fistula in arch of superior canal, recovery.

A girl, age eight years, was admitted eight weeks after a simple right mastoidectomy had been performed. Inquiry revealed that her mastoid process had been highly pneumatized. Since a time three weeks after the mastoidectomy she had been suffering with right frontal headaches which were more severe at night. At the same time her temperature ranged between 99 and 101. She refused to play and preferred to stay in bed. At the time of admission, February 19, 1935, there was a thick whitish purulent discharge coming from the lower angle of the mastoid wound while the canal presented a pulsating discharge. There was a questionable involvement of the lower segments of the facial nerve. X-ray of the petrous bone on the third day after admission revealed a complete absence of structure of the apex cell area. The left petrous apex tip was cellular and well pneumatized (see Figs. 5 and 6). Because of the headache, the persistent discharge for eight weeks after opera-



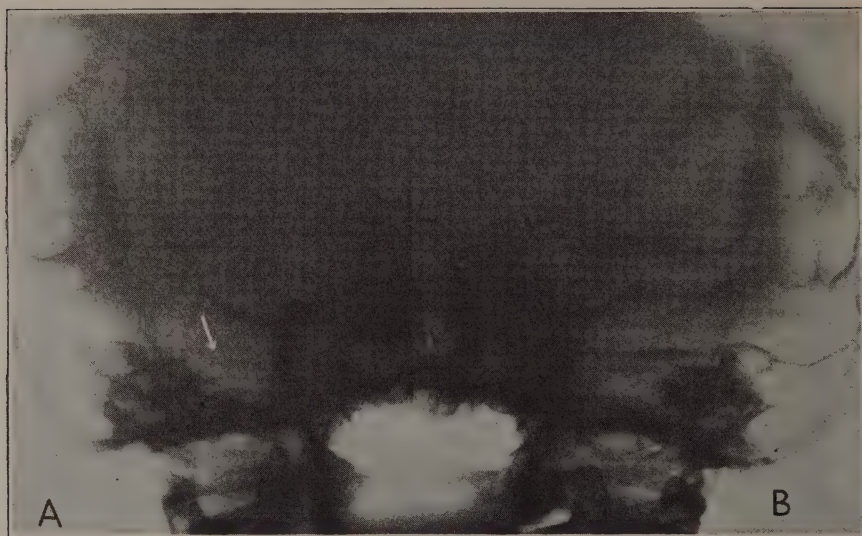


FIG. 5 (*Case 8*). Roentgenogram taken before the operation. Note complete loss of outline and definition of the area of the apex tip as compared with that on the uninvolved side. The patient was in the Stenver position.

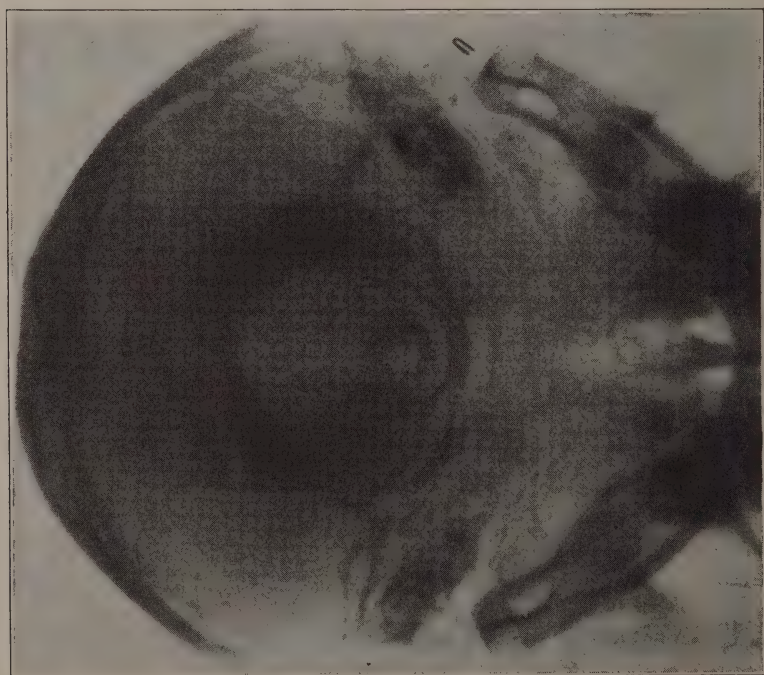


FIG. 6 (*Case 8*). Roentgenogram taken with the patient in the Taylor position, ten days after operation. Note the complete loss of definition of the area of the apex tip.



tion, the very definite x-ray evidence, and the temperature, a petrous exploration was performed.

Operation consisted of removing the bone down to the superior semicircular canal and deep into Trautmann's triangle. Some discolored granulations were found which led into the arch of the superior canal. A probe inserted here fell into the apex area. Withdrawal of the probe was followed by a release of a sufficient quantity of pus to fill the mastoid cavity, approximately two drams. The tract was enlarged by gentle curettage and some strands of silkworm gut were inserted as a drain. The enlarged fistula was a little more than 3 mm. in diameter. The dura was elevated to expose the apex area in order to search for a possible epidural abscess. The middle meningeal artery was clamped by means of two clips and divided between them to insure hemostasis. No epidural abscess was found.

The headache was no longer present on the day following the operation. Her condition continued good until her discharge from the hospital a little more than a month after the finding of the fistula, when she was discharged as fully recovered.

*Summary.*—February 19. Admitted: Mastoidectomy eight weeks ago. Temperature and frontal headache for five weeks.

February 22. Second operation, because of headache and positive x-ray finding, in addition the continued discharge and duration. Fistula found in arch of superior canal. Two drams of pus evacuated.

March 25. Recovery uneventful. Discharged.

#### COMMENT

A case in which a severe nocturnal headache persisted for five weeks after a mastoidectomy had been performed three weeks previously. A most striking and unequivocal x-ray finding of destruction was furnished in this case.

#### SUMMARY AND CONCLUSIONS

A comprehensive review of the subject of petrous pyramid suppuration has been presented to which has been added the results of the studies and experiences of the authors. A consideration of the symptomatology and clinical phases is featured. The importance of fistulæ in the pathogenesis and the treatment of this condition is stressed and established. Fifty-three fistulæ were collected from the literature which had been observed at autopsy or at operation. These fistulæ were located at nine different sites in the middle ear and mastoid cavity. They have been tabulated according to location and according to the authors who reported them. Six cases of multiple fistulæ were included. In addition nine retropharyngeal abscesses which drained an empyema of the petrous apex are included.

Eight clinical experiences are related of which six terminated in recovery.

An attempt has been made to simplify and clarify the indications for operative interference and a plan of surgical attack is offered.

## DISCUSSION

P. E. MELTZER, M.D., Boston, Mass.: My discussion of Doctor Myerson's paper will be chiefly from an anatomical and pathological viewpoint. My remarks dealing with gross anatomy are based on my own dissections of the temporal bone, and though not arranged and classified as systematically as Doctor Myerson's, they are, nevertheless, the result of many dissections. Such remarks as I may make dealing with the pathology are made by inference only as I have no pathological material to qualify any statements I make. However, as we are dealing with types of cells and bone structure very much the same as that encountered in the mastoid process, and as these cells are contiguous, I believe that the pathology when present will be the same, *i.e.*, simple congestive or hemorrhagic changes, granulation with or without suppuration and with or without bone absorption, and finally bone necrosis with formation of pus.

### THE ANATOMY OF THE PETROUS AREA

If one makes a horizontal section anteroposteriorly from the tip to the mastoid bisecting the middle ear and labyrinth, one would immediately recognize that an area was exposed which only the most qualified otologist is justified in entering surgically. Without anatomical knowledge of this particular portion of the temporal bone proper evaluation of symptoms and certainly the surgical approach is impossible. From what I have seen of the cellular distribution in the region of the antrum it is my opinion that this route is by far the commonest pathway of infection into the petrosa.

Obviously, we do not see petrositis in very young children, particularly under one year of age, because pneumatization has not yet made great headway. When it does occur in young children I believe that the pathway is along the perivascular and perineural spaces into the marrow of the petrous. Throughout life I believe this still may be one of the pathways by which this area becomes infected. As pneumatization goes on in the course of the development of the individual the petrous area as a rule does not keep pace with the pneumatization which extends from the region of the antrum into the developing mastoid.

This is not the time to discuss the reasons why this does not occur. In this respect Myerson's figures in regard to the cellular development of the petrous bone are in accord with the majority of investigators, namely, that about 70 percent are not pneumatized.

Considering the pathological changes which occur in this region it is reasonable to assume that because of the continuity and contiguity of the paralyrinthine cells with the middle ear and antral cells, any severe inflammation is likely to involve these cells of the petrosa just as it is likely to involve the cells of the mastoid region. The extent of the involvement may be simple acute or passive congestion or a marked acute congestion as seen in early mastoiditis. It may go on to a point where granulations are formed with or without bone absorption and with or without suppuration. It is the unusual

case, because of factors which cannot be definitely determined, that goes on to an isolated localized suppuration or a diffusing osteomyelitis.

The roentgenologist must constantly keep in mind the anatomical variation in the arrangement as well as the type of cells in this region, otherwise, if he looks for the same character of cell in the petrosa as he finds in the mastoid he is likely to be misguided.

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## SOME CONSIDERATIONS OF ADENOID BLEEDING

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There are few events in the daily life of the laryngologist that cause more annoyance than the occasional telephone call, from three to six hours following an uncomplicated operation for the removal of a child's tonsils and adenoids, announcing that bright blood is trickling from the nostril or has been vomited in appreciable quantity. This event is sufficiently uncommon to warrant the feeling that it should never occur; it is potentially serious enough to brook no delay in its management; and its occurrence is often unexplainable by any facts which can be obtained by local examination. Not the least unpleasant aspect of the situation is the necessity of explanation to the parents as to why the bleeding has occurred or why it is necessary to return the child to the operating room, with or without a second anesthesia, for its control. If, in the case of adenoid bleeding, postnasal packing is deemed necessary, there is the possibility of a middle ear complication. Intravenous saline infusions may be needed. Nevertheless, to neglect obvious active postoperative bleeding with optimistic nursing orders to apply an ice collar, give a hypodermic injection of morphine and watch the pulse is, in my experience, to put an unwarranted trust in Providence that may unexpectedly lead to a tragedy than which there is no greater in all surgery.

There may be operators who can look back over a long and busy career and fail to recall any instance of alarming postoperative adenoid bleeding, even in a large number of cases. For those men, however, who are responsible for the conduct of a hospital clinic, in which the work must inevitably be done by a changing personnel and to some extent by a less experienced house staff, albeit under competent supervision, there will inevitably occur a certain number of cases of postoperative bleeding, requiring prompt control.

For this reason it has seemed to me valuable to make some analysis of this work in a large hospital clinic with a view to discovering if possible why and under what circumstances this complication occurs and by what means it may be lessened or entirely eradicated. I shall in the main direct the inquiry into the occurrence of bleeding from the adenoid to such extent as requires, in the judgment of the surgeon in charge, insertion of a postnasal pack. To this end I have gathered the figures summarized in Chart I.

CHART I.—STATISTICS OF POSTOPERATIVE TONSIL AND ADENOID BLEEDING

<i>Operators</i>	<i>Years</i>	<i>Number of operations</i>	<i>Total number of hemorrhages</i>	<i>Tonsil bleeders</i>	<i>Adenoid bleeders</i>	<i>Total percent of bleeding</i>	<i>Tonsil %</i>	<i>Adenoid %</i>
A. O. P. D. Clinic.....	1928-1934	10,745	294	84	210	2.7%	0.79%	2.0%
B. O. P. D. Clinic.....	1930-1934	6,145	132	35	97	2.14%	0.57%	1.6%
C. 20 House-Officers...	1930-1934	3,116	60	13	47	1.95%	0.4%	1.5%
D. Clinic— 11 visiting men....	1930-1934	3,029	72	22	50	2.37%	0.7%	1.65%
E. Private Ward (Entire staff of 30)	1928-1934	5,216	32	9	23	0.6%	0.17%	0.44%
F. 15 private operators.	1928-1934	2,923 56%	32	9	23	1.09%	0.3%	0.79%
G. 15 private operators.	1928-1934	2,293 44%	0	0	0	0	0	0
H. Manhattan E. & E...	1930-1932	13,344	549	..	..	4.0%	..	..
I. Manhattan E. & E... (12 operators)....	May, 1931- Nov., 1931	6,370	246	144	102	3.8%	2.2%	1.6%



Chart I. In line A are the figures obtained from the Out-Patient Department Tonsil and Adenoid Clinic in the Children's Hospital in Boston, for the six years, 1928 to 1934. In 10,745 operations there were listed 294 instances of postoperative bleeding, 84 from the tonsil, 210 from the adenoid, a total incidence of 2.7 percent and of 2.0 percent for the adenoid. It will be noted throughout the chart that in every instance, except in the comparative figures from the Manhattan Eye and Ear Infirmary, the adenoid bleeding was always more than twice as frequent as that from the tonsil. The reason for this is not at once apparent. In this series of cases the adenoid was removed last, all tonsil bleeding being entirely checked before adenoidectomy. The customary practice of suturing suspicious vessels in the tonsil fossa doubtless had something to do with this proportion. A total percentage of 2.7 percent seems unduly high and certainly arouses one's curiosity as to its cause and its remedy. I feel that one outstanding factor was contributory. In the years 1928-1930, the clinic arrangements were such as to make it necessary for the two operators, having finished the operative work, to transfer their activities to a frequently understaffed outpatient clinic. This situation, no matter how conscientious the men, resulted in the introduction of a certain sense of pressure and haste in the operative work which I believe had a distinct bearing on the postoperative bleeding here under consideration. Each man was required to perform from six to seven operations, a small number when judged by the output of some clinics, but still a sufficient one to introduce a sense of need for completing the work as soon as possible before repairing to the outpatient clinic. No man can do his best work under these circumstances.

For this reason, modifications were made in the system to the extent that it no longer was necessary for the operators to undertake the outpatient work on the same afternoon, other help being provided for this task. The benefits of this change I believe are reflected in the figures in line B, taken for the years 1930-1934, when the improvement was inaugurated. Here the bleeding percentage in 6,145 cases is reduced from 2.7 to 2.14 percent, a figure, however, still leaving much to be desired. During these four years the system called for operating by two distinct classes of operators: (1) a group of eleven well trained and thoroughly experienced visiting surgeons who came in on one afternoon a week, performing their six or seven operations without further clinical duties; (2) a group of twenty house officers, as adequately trained under competent supervision as possible, but naturally lacking in the years of experience possessed by the older

group. At any one session a visiting man and a house officer worked side by side simultaneously. In view of this arrangement, the figures in lines C and D are most interesting. It will be noted that, in an almost equal number of operations the house officers (line C) had a total bleeding percentage of 1.95 percent, while the more experienced visiting men (line D) had a figure of 2.37 percent. Separate figures for tonsil and adenoid bleeding corresponded. How can this paradox be explained? Here again I believe the personal equation is the predominating factor. The attitude of these two groups is necessarily different. For the visiting surgeons, the procedure is a routine one, previously performed numberless times and to some extent a chore. To the house officer it is a relatively new experience to which he is apt to devote an intense, albeit less expert, interest. Hence, it comes about that the busier man with his outside interests and problems comes inevitably to take toward the work an attitude of a task to be performed into which creeps, unconsciously, an element of haste which is absent in the case of the less experienced man, unless he likewise becomes inoculated by the same germ through contact. Haste in this way leads, I believe, to a higher percentage of postoperative bleeding.

It becomes still more evident that this element of pressure is an important factor when we examine the record of a group of visiting surgeons in care of their own private patients. Here a man operates on one or at most two or three patients at a sitting. In many instances this group was made up of the same men working in the clinic and this affords a direct comparison between the two situations. In line E are seen the figures for the Private Ward staff of thirty men, who operated on 5,216 patients with a total bleeding percentage of only 0.6 percent, as compared to 2.37 percent in the clinic. Moreover, 56 percent, or 2,923 of these cases, were operated upon by fifteen men (line F) amongst whom all the bleeding occurred, and whose percentage was thus 1.09. Two thousand, two hundred ninety-three cases were operated upon by the remaining fifteen men of the private staff (line G) without any recorded postoperative bleeding whatever. How may this difference be explained? In the one instance, in which a man working both privately and in the clinic did a fairly equivalent group of operations, his bleeding percentage in the clinic was 2.0 percent, in the private ward 1.0 percent. In both groups the preoperative care and examination were, if anything, better in the case of the clinic patients and the etherization and postoperative care equally good. I can only fall back on the difference in mental attitude incident to the necessity of accomplishing a greater amount of work in a given space of time. Yet even here, one may adduce figures to refute

this explanation. From an analysis of the clinic figures for six years, above, it appears that the average highest monthly bleeding percentage (5 percent) occurred in an average of 124 operations per month. The lowest corresponding percentage (0.5) was based on an average of 112 operations. In other words, there was ten times as much bleeding, although the average number of operations was only very slightly increased. This does not look as though the pressure of doing a large volume of work raised the bleeding percentage. Looked at from a different angle, it appears that the average of the largest number of cases per month (184) showed a bleeding of 2 percent, while the average of the smallest number of cases per month (81) showed exactly the same (2 percent). It would be hard here to maintain that the busiest months are those of the greatest postoperative bleeding. The worst monthly figure, a postoperative bleeding percentage of 6.7, was in October, 1929, the best (1.5 percent) in November, 1932.

More recently the clinic system has again been changed, and the operating is now done entirely by the house staff, with a resident surgeon in charge. Neither of the two operators, therefore, now has any extra-hospital duties and it has been interesting and gratifying to note a still lower percentage of postoperative bleeding following this change.

For the sake of some check on these figures, I have added, in lines H and I figures from the Manhattan Eye and Ear Infirmary. In a total of 13,344 cases which, unlike our own, included adults, the bleeding percentage for two years was 4.0. A group of twelve operators (line I), working in the Manhattan Clinic for six months, had a percentage of 3.8 (2.2 for the tonsils and 1.6 for the adenoids, a reversed relation as compared with our own figures).

Granted that postoperative bleeding is to a greater or less degree an inevitable complication in a tonsil and adenoid clinic, what is its basic cause and can anything be done to avoid it? In an effort to throw some light on this question, I studied carefully certain factors in 200 clinic operations for the removal of adenoids, practically all in conjunction with tonsillectomy.

In view of the above mentioned fact that adenoid bleeding occurred more than twice as frequently as did tonsil bleeding, it is only the former which is here considered. The group of 200 was divided into two series. Series I, of 100 cases, was treated as follows: Tonsillectomy was first completed and a dry fossa assured before the adenoid was removed. Following adenoid removal, by the use of sponge tampons in the nasopharynx, the bleeding was checked until

direct inspection of the pharynx showed complete cessation of bleeding. At the same time all blood loss from the adenoid, either through the nose or saturating the sponges, was measured by weight. The weight of the adenoid and the time before the nasopharynx was found to be dry by direct inspection, were also recorded. The patient was then returned to bed and the throat inspected at hourly intervals for any signs of adenoid bleeding.

Various authors, in writing on the subject of adenoid removal, have stressed the importance of the principle here carried out in Series I, namely, the inadvisability of returning a patient to bed until the bleeding from the adenoid had entirely ceased. Surgically, this seems an obviously sound principle, comparable to a similar situation in any other part of the body. It affords opportunity, by elevation of the soft palate, either by an elevator or, as Beck advocates, with a catheter retractor, to inspect the pharyngeal wound itself and carefully search for tags and remnants, and would seem to assure the operator the greatest protection against subsequent hemorrhage. It does, however, require a somewhat more prolonged anesthesia, about which more will be said below.

In Series II an entirely different procedure was carried out. Following removal of the adenoid and before cessation of bleeding therefrom, the patient was taken back to bed, hung over the side of the latter and allowed to bleed into a basin till all bleeding ceased. This blood was likewise measured by weight as was the adenoid mass and the time until all bleeding in this position ceased.

Of these two procedures, it would seem that that of Series I was more painstaking and more likely to insure against subsequent bleeding. In Series II there was no inspection of the nasopharynx beyond careful palpation for adenoid tags. The anesthesia, however, was shorter, since no additional ether was required to abolish the throat reflexes in order to permit a tranquil and complete inspection of the nasopharynx. By the same token, the patient was returned to bed in a shorter time, since there was no waiting for the immediate adenoid bleeding to cease.

This apparently somewhat lax method of Series II was adopted primarily because it seemed to be the policy of certain members of the private ward staff whose records as to postoperative bleeding were by far the best of all the visiting surgeons. It was formerly the custom of the author to assure himself of a dry nasopharynx before any case left the operating room. A series of three bleeding cases in one week led him to inquire into the methods of other operators and to discover that this method of Series II was a routine practice with



the majority of the other men. Hence its adoption as a part of this study.

In addition, all of the cases in each series had a preliminary clotting and bleeding time.

Chart II gives a general view of the results in these two series. In analyzing the individual items, a number of interesting facts come to light. The general bleeding time in the two series showed little variation. The time of operative procedure measured from the start of the tonsillectomy to the finish of the adenoidectomy varied considerably in the two series, being more than half again as long in the first as in the second. This is hard to explain, since the work was done by a group of six or seven men, whose time should average about the same. However, a large proportion of the cases was done by house officers whose technic naturally improved with experience. The average duration of the bleeding from the adenoid varied but little (four to six minutes) in spite of the different technic adopted in the two series, and, most interesting of all, the total blood loss in the two series was almost identical. There was some variation in the weight of the adenoid removed. In the first series postoperative packing was required in four instances, while in six cases after signs of bleeding the oozing subsided spontaneously. In the second series three cases required packing, while none of the others showed signs of suspicious bleeding.

It is clear from these figures that there was little to choose between these two methods, in respect to the amount of blood loss or the likelihood of postoperative bleeding severe enough to require packing. If anything, the balance is in favor of the less painstaking procedure. Can one, then, from these figures, gain any knowledge as to the type of case that is likely to give trouble or to require postoperative packing? Can one, in other words, by examination of this data, detect warnings which, if heeded, might enable one to forestall trouble?

Let us first examine the figures on those patients who required postoperative packing in the two series. The comparison between the two is shown in Chart III.

These figures, alas, give little clue as to why these seven cases required postoperative packing. Beyond the somewhat excessive bleeding of 80 grams (2.6) oz. in Series II, there are no figures which differ essentially from the normal average for all the cases. So far as their statistical characteristics are concerned, these seven cases differed but little from all the others which showed no tendency to postoperative bleeding. Jones, in his report of the Manhattan Clinic,

CHART II.—COMPARATIVE TABLE OF 200 CASES OF ADENOIDECTOMY

Series I. Bleeding entirely checked before patient left operating room  
 Series II. Patient sent immediately to ward after adenoïdectomy

Series	General bleeding time	Time of operation	Duration of adenoïd bleeding	Adenoïd weight	Weight of blood loss	Postoperative bleeding Packed	Not packed
I. (100 cases)	Average 3 minutes Shortest 0.5 Longest 9.0	Average 13 minutes Longest 48 min. Shortest 6 min.	Average 4 minutes Longest 20 min. Shortest 1 min.	Average 2.5 grams Largest 4.6 Smallest 0.5	Average 52 grams Largest 1,529 Smallest 49	4 cases	6 cases
II. (100 cases)	Average 2 minutes Shortest 1.0 Longest 10.0	Average 8 minutes Longest 19 min. Shortest 5 min.	Average 6 minutes Longest 15 min. Shortest 1 min.	Average 1.5 grams Largest 4.59 Smallest 0.25	Average 50 grams Largest 1,509 Smallest 89	3	0
Total average	2.5 minutes	12.5 minutes	5 minutes	2 grams	51 grams	Bleeding percentage, 3.5	

CHART III.—BLEEDING REQUIRING POSTOPERATIVE PACKING

Series	Number of cases	Av. operation time	Average ad. bleeding time	Average blood loss	Systemic bleeding time (Av.)	Average weight of adenoïd
I.	3	15 minutes General average 17 min.	4.1 minutes General average 4 min.	50 grams General average 52	4.3 minutes General average 3	2.2 grams General average 2.5
II.	4	10 minutes General average 8	8 minutes General average 6	80 grams General average 50	2.5 minutes General average 2.0	1.1 grams General average 1.5

states that 90 percent of their postoperative adenoid bleeding was checked by the removal of adenoid remains. In only two of the seven cases here summarized as requiring packing were any adenoid remains found. This fact makes it the more desirable to discover, if possible, the etiology of this complication.

With a view to determining the relationship, if any, between the various factors here recorded, certain checks were carried out between these factors:

1. Relationship between a prolonged immediate adenoid bleeding, the amount of blood involved in this bleeding and the occurrence of postoperative bleeding (requiring packing) or temporary oozing was observed. It was thought that there might be derived here something of diagnostic or prognostic value. The results are shown in Chart IV.

CHART IV.—RELATIONSHIP BETWEEN PROLONGED BLEEDING, BLOOD LOSS AND POSTOPERATIVE BLEEDING

<i>Average of ten longest bleeding times</i>	<i>General bleeding time</i>	<i>Average blood loss</i>	<i>Packed</i>	<i>Oozing</i>
14.7 minutes	3.9 minutes	74.3 grams	1	1
Average for 200 cases	Average in 200 cases	Average, 200 cases	Total in 200 cases	Total in 200 cases
5.0 minutes	2.5 minutes	51. grams	7	6

From Chart IV it will be seen that in ten cases with an average immediate adenoid bleeding time three times the average for the whole group, only one case required packing and only one showed temporary oozing. In this group of ten the general bleeding time was somewhat elevated (3.9 minutes) over the general average (2.5 minutes), a very natural relationship. Also, quite naturally, the total average blood loss of 74.3 grams was considerably above the average (51 grams) and yet the postoperative complication does not appear to any real extent within this group.

2. This same situation was viewed from another angle by taking the ten largest amounts of immediate blood loss and noting their relation to the adenoid bleeding time and the occurrence of postoperative bleeding. This is shown in Chart V.

Here again, in spite of an average blood loss twice the average for the series, there is almost no tendency for the postoperative bleeding to occur within this group. The greater blood loss corresponds, as one might expect, with the higher general bleeding time, and an

increased adenoid bleeding time, but the postoperative situation fails to follow suit.

CHART V.—RELATIONSHIP BETWEEN BLOOD LOSS, ADENOID BLEEDING TIME AND POSTOPERATIVE BLEEDING

<i>Average ten largest blood losses</i>	<i>General bleed- ing time</i>	<i>Average adenoid bleed- ing time</i>	<i>Packed</i>	<i>Oozing</i>
109 grams	3.3 minutes	9 minutes	1	1
Average for 200 cases	Average for 200 cases	Average for 200 cases	Total in 200 cases	Total in 200 cases
51 grams	2.5 minutes	5 minutes	7	6

3. There was a direct correlation between the size of the adenoid and the immediate blood loss attending its removal. The average weight of the ten largest adenoids, 4 grams (twice the normal average), was associated with an average blood loss of 70 grams (51 average for the 200 cases). The ten smallest adenoids (average 0.4 gram) produced an average blood loss of 33 grams. No postoperative bleeding occurred in either of these groups.

4. The question arose as to whether prolonged time of operation (and, therefore, of anesthesia) might have some bearing in the matter. The average of the ten longest operative times (26 minutes—average 13) was associated with an average adenoid bleeding time of seven minutes, five being the average for all the cases. No postoperative bleeding occurred in this group. On the other hand, perhaps refuting my suggestion that "haste makes waste," there were no bleeding complications among the operations done in the shortest times.

5. Finally, the ten cases showing a greatly increased general bleeding time were checked in respect to the duration of adenoid bleeding and tendency to postoperative bleeding. The average time in these ten was a bleeding time of 7.3 minutes, as against an average for the whole group of 2.5 minutes. Yet these cases showed an average adenoid bleeding time of only 6.3 minutes, 5 being the group average. Among these ten cases there occurred only one case of postoperative bleeding requiring packing.

Why, then, does postoperative bleeding occur? So far as our figures go, it is essentially independent of a prolonged general bleeding time, an unduly prolonged operation, a prolonged and increased immediate loss of blood and an excessively large adenoid. There remains one factor as yet only hinted at and incapable of definite statistical proof, which I believe may nevertheless contain the answer



to the problem, namely, the depth of anesthesia. To thoroughly understand this aspect of the matter it is necessary to consider in some detail the question of anesthesia in respect to tonsillectomy and adenoidectomy.

Since the earliest history of the tonsil and adenoid operation there has been a controversy over the question of anesthesia. Before the present days of perfection in the administration of ether some of the most eminent laryngologists expressed a fear of its use and preferred to operate, at least in the younger children, without any anesthesia at all. This same, and from the modern point of view, rather inhumane attitude, has been the rule in Europe up to within the past ten years. It has been argued in behalf of this attitude that the child suffered more from the shock and fright of the administration of the anesthesia than from the pain of the operation. This can scarcely be urged today in view of the almost routinely tranquil gas-ether sequence so deftly administered by the trained anesthetist. In the year 1900 several of the most eminent members of this Society expressed themselves as in favor of adenoidectomy in children without anesthesia, even if under these circumstances it was found necessary to do the operation piecemeal at several sittings. Fatalities attributable to general anesthesia were reported with disquieting frequency. All admitted that the work without anesthesia required a deft sense of touch and dextrous manipulation of the forceps or curette, and yet we read of no reports of annoying postoperative bleeding by these men. Kimball, writing in the Transactions of this Society for 1900 on "Adenoids from the Standpoint of Hemorrhage," says that he is inclined to treat the matter as a surgical bugaboo, though it is to be noted that in twelve years he performed only 350 adenoidectomies. He decried advances in technic which hastened the time of operation and concluded that postoperative bleeding was proportionate to the rapidity of operation.

Of late years more men have advocated a complete surgical anesthesia, primarily in order that the work may be carried out with greater facility of careful inspection of the nasopharynx for any adenoid remnants. All writers agree unanimously on the point that adenoid remains predispose to postoperative bleeding. Offhand it would seem that the more active the throat reflexes the more difficult would be the attainment of a clean pharyngeal wall, and the inspection of the adenoid bed for tags and remnants. The further objection has been offered that through muscular contractions portions of the nasopharynx, other than the adenoid tissue, might be incidentally included within the range of the adenotome. And yet, as pointed

out above, the advocates of the lightest form of anesthesia—that is, none at all—seem not to have been troubled by postoperative bleeding. It may be well that the initial bleeding from the cut pharyngeal vessels is more effectively checked when the pharyngeal reflexes and muscles are more active than when they are more profoundly relaxed as in the case of a deeper anesthesia. Under the latter, the throat may appear dry when the patient leaves the operating room, but bleeding may start up again when the reflexes return. The author, for several years an advocate of complete pharyngeal relaxation during adenoidectomy, has, because of several instances of postoperative bleeding, changed to a technic which delays adenoidectomy until a complete return of the pharyngeal reflex, with a marked diminution in this complication. This same method is rigidly adhered to by several men whose bleeding percentages are the lowest found in the figures above cited. In this connection it is extremely interesting to note that in our entire series of cases, postoperative bleeding was practically unknown in those patients on whom adenoidectomy alone was performed. One wonders whether the lighter anesthesia usually incident to the shorter procedure has any bearing on this fact.

The method of removing the adenoid must be considered to have some effect on the liability to subsequent hemorrhage. In the earliest days of laryngology it was customary to employ a curved, biting forceps (Löwenberg) which, introduced behind the soft palate, grasped the adenoid mass more or less completely and literally wrenched it from its bed. Apparently, in spite of the crudity of this procedure, subsequent bleeding was not a particularly frequent complication, though it is difficult to understand why remnants were not often left behind. Even today one occasionally sees this instrument competently used by men of the old school, somewhat to the perplexity and horror of the younger generation.

More commonly in use are two types of instruments, the curette in its various forms and the adenotome. Each has its devotees, the former particularly in the West and South. A combination of the two methods is popular, the curette being used to remove small tags of adenoid tissue left behind by the adenotome. Jones found a greater tendency to postoperative bleeding with this combination and in his clinic at the Manhattan Eye and Ear Hospital now uses the adenotome exclusively, introducing it once in the midline, and again on either side of the nasopharynx. More recently a single instrument has been devised which combines the features of both types of instrument.

In most clinics it is customary to remove the tonsil first and then the adenoid. Some men, however, advocate preliminary removal

of the adenoid, then the insertion of a gauze pack in the nasopharynx, left during the period of tonsillectomy. Jones states that this procedure has in the last 7,000 operations done at the Manhattan materially reduced the postoperative bleeding.

Various other factors must necessarily play some part. Any case of blood dyscrasia or true hemophilia or unduly long bleeding or clotting time is a dangerous case to operate upon and one in which trouble may be expected. Any technic in which the underlying muscle is invaded will be a potential case for postoperative bleeding. The presence of pharyngeal infection predisposes to similar trouble.

In the author's experience the best results and the fewest complications come from a careful preoperative investigation of the patient, the employment of a competent anesthetist, who knows at all times the depth of the anesthesia, a careful technic carried out with neither haste nor delay, and assurance of returning pharyngeal reflexes before removal of the adenoid.

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MEETING OF THE MIDDLE SECTION HELD IN INDIAN-  
APOLIS, INDIANA, JANUARY 23, 1935, UNDER THE  
CHAIRMANSHIP OF DANIEL W. LAYMAN OF IN-  
DIANAPOLIS, INDIANA.

A CLINICAL STUDY OF THE FUNCTIONING OF THE  
MAXILLARY SINUS MUCOSA

By EDWARD KING, M.D.

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The function of the sinus mucosa as demonstrated by Hilding, Proetz and others in experimental work on dogs, is well known to all rhinologists. Some time ago Hilding made a clinical observation on a patient who had a large intranasal window in the antrum. He put India ink in the antrum and by means of a nasopharyngoscope watched the flow of the ink past the artificial opening out through the natural orifice.

Recently we published a series of cases studied by means of x-ray with the aid of iodized oil before and after the intranasal antrum operation. In connection with this study a few cases were reported in which an attempt was made to determine whether the drainage from the antrum occurred through the artificial opening or through the natural orifice.

Since then some more cases have been examined and the conclusions are the same as published in the previous publication.

The technic consisted in placing a small quantity, 1 c.c. or less, of iodized oil in the antrum in which an artificial opening had been made and observing the flow of the oil out of the antrum by means of a series of x-ray pictures.

In the original communication seven cases were studied. Since then five more cases have been examined. In these last five cases the examination was conducted about three or four weeks after the operation, before the opening had an opportunity to contract. In spite of the large opening the tendency of the oil was to flow out through the natural orifice, although some was found draining by gravity through the artificial opening.

In the original paper two cases were reported in which the oil did not move after a long period of observation (ten hours). Both of these cases had been operated upon successfully by the intranasal



route, *i.e.*, the symptoms and discharge disappeared and x-rays showed no filling defect in the antrum. It was not understood at that time why the oil did not move. Since then both patients have been under treatment for antrum infection, which recurred almost simultaneously with an acute cold in the head. Now we feel that the infection was dormant and the cilia inactive because of the latent infection.

We studied a third case in which the intranasal operation was performed eight years ago. This patient still has a good-sized opening, but every time she catches cold it is necessary to wash the antrum frequently. The history showed that following the intranasal operation the antrum infection did not clear up until strong solutions of silver nitrate had been instilled into the antrum six or seven times. The functional test with iodized oil showed no movement of the oil in spite of the fact that the x-ray picture showed a perfect filling.

The conclusion from this case is that the strong silver nitrate solutions destroyed the cilia and the mucosa no longer functions normally.

We have two other cases in which the intranasal operation was performed successfully and in whom the functional test showed no movement of the oil. These cases are still under observation. They have had no return of symptoms, but we feel that the antrum is still diseased and may flare up at any time.

Hasty, at a meeting in Cincinnati, showed a case in which the filling defect was marked. After the use of iodochlorol a few times the swelling disappeared and the antrum mucosa looked normal. This was surprising because it was my opinion as well as that of a number of other laryngologists that when a pronounced filling defect is found in a chronic infection of the antrum the radical operation is indicated.

The treatment of the chronic antrum infection can be carried out successfully in some cases. We have three cases in which the filling defect was very marked. My own case illustrates that point. We have two others in which iodochlorol was used exclusively over a period of a few months and the mucosa returned to its normal size. In both of the cases the functional test was made to determine whether the cilia were behaving properly. In one the function was prompt and efficient and in the other the oil did not move. In the latter case the patient returned after some months with a recurrence of the infection and a pronounced filling defect. In my own case the function is normal.

It is our opinion that we can determine whether the antral mucosa functions properly by instilling a small quantity of iodized oil into the antrum and watching its action by means of a series of x-rays. The benefits of the intranasal operation may be determined by the same method.

## DISCUSSION

HORACE R. LYONS, M.D., Chicago, Ill.: The study of the physiology of the antral mucosa by microscopic study, direct examination with an antrascopes, the use of opaque oils and the x-ray have given clinical data which greatly increases our understanding of infections in the maxillary sinus.

The study of chronic maxillary sinusitis today consists of first, a thorough and complete history which includes an allergic survey; second, clinical observation over a reasonable length of time; third, physiological study which includes irrigation to obtain a view of the type of discharge and for bacterial examination, x-ray study which, after observation, may be repeated with the use of an opaque fluid to visualize a thickened membrane, a filling defect or ciliary activity; fourth, when necessary the use of an antrascopes actually to visualize the interior of the sinus; fifth, allergic study of an exhaustive character before radical operation is advised. Allergy is so important today that it is not difficult to see into the near future when improved tests and treatment may supplant our present treatment of the majority of sinus infections.

Doctor King, in his previous report, and again today has illustrated a physiological test for ciliary activity within the antrum. It occurs to me that the value of this test would be in obtaining identical results by repeated tests on the same individual. For instance, given a chronic infection of the antrum in which this test showed no ciliary activity, it would be more conclusive if there were repeated tests with identical results. It may prove that the cases which have some ciliary movement on repeated tests may be cured without denuding the membrane by external approach, while those that, on repeated tests, never show any ciliary activity require a radical operation.

The value of this test, it seems, would be in correlating the results of repeated tests with other clinical data.

WALTER H. THEOBALD, M.D., Chicago, Ill.: King's paper brings out some very interesting clinical observations. It is quite evident from his report and Hilding's, as well as a recent publication by Latta and Schall of Omaha, that regeneration of epithelium within the antrum apparently occurs. The latter are of the opinion that undifferentiated basal cells, which are not involved in any severe degenerative process possess potentialities of forming either ciliated or goblet cells. With this histological observation in mind, the method described by Doctor King provides a functional test by means of which the behavior and presence of this ciliated epithelium within the antrum may be determined either during a chronic diseased condition or following the radical operation. In the latter type of cases one is not assured always of regeneration of ciliated cells. Their presence has been observed histologically in only a few cases where the mucosa was removed one month or more after operation.

In lieu of this demonstration of postoperative cases, we considered that in like manner one might study the ciliary activity of a known normal maxillary sinus

and thereby note the rapidity with which a radiopaque substance is removed from the antrum. For this purpose we were of the opinion that the use of barium as a radiopaque substance might provide a more nearly normal physiological response from the cilia because of the heavy viscosity of lipiodol. Therefore, one c.c. of a heavy mixture of barium sulphate in water was injected into the antrum through the natural ostium without the use of cocain or other medication that might depress or stimulate ciliary activity. The study of these x-ray films revealed a prompt migration of barium in the direction of the natural opening within twenty-five minutes after instillation. Further exposures were made on succeeding hours and days and the lapse of time required for the entire removal of barium was about eight days. A small residue was noted making its way up the side of the antrum on the sixth day. On the eighth day the sinus was found to be nearly empty. Nineteen hours after injection, a very clear pathway of barium is shown reaching up to the natural opening and making its way toward the posterior nasal space.

In another case which I am presenting of a previous radical operation on the antrum three and one-half years ago, this functional test revealed only a small diminution of barium after two days and nineteen hours. Another exposure nine days later showed only a slight decrease in the amount of barium as contrasted with the case reported above. It would appear from this observation that ciliary function of the antral mucosa was absent. And yet there has been no recurrence of infection in this case since the operation three and one-half years ago.

DANIEL W. LAYMAN, M.D., Indianapolis, Ind.: The efficacy of our treatment of diseased conditions, especially of the accessory nasal sinuses, is in direct ratio to our knowledge and appreciation of the function of these cavities. A great deal of literature has appeared, during the past few years, dealing with the physiological function of the mucous membrane, ciliary activity, that mysterious condition known as allergy, and the effects of chemicals and biochemical substances on nasal ciliated epithelium. Some of these papers and clinical reports were written by members of this Society; I refer to Proetz, Fenton, Lierle, Mullin, Grove and others, and to this list I now add Doctor King's name. All have contributed to our knowledge and appreciation of the function of the mucous membrane of the nasal cavity.

What has this newer knowledge of the function of the nasal and sinus mucosa accomplished? It has necessitated changes in the method and kind of treatment of the diseases of the nasal cavity, and these changes when approved by the medical profession will place our therapeutic efforts on a more rational basis. It has brought about a better agreement and accord among the rhinologists in their interpretation of pathological symptoms and as to the operative procedure indicated. Consequently, the marked diversity of opinion which existed between the conservative and the radical rhinologists is disappearing. A conservative trend is noticeable, that is, operative procedures have receded into the background since this better understanding of the many influences which tend to undermine the natural resistance of the mucosa.

However, a certain amount of diversity of opinion among physicians will always exist, due probably to the fact that physicians differ in their qualifications as diagnosticians, in surgical judgment, and its surgical application.

This recalls a remark that Dr. Joseph Bryant, a New York surgeon, made, when I was an interne in a New York hospital. When he was asked whom he would select as his surgeon if a major operation had to be performed on him, his reply was: "I would want Dr. Charles McBurney to make the diagnosis, Dr. Wm. T. Bull's surgical judgment as to the kind and extent of operation and Dr. Robert Abbe to perform the operation."

EDWARD KING, M.D., Cincinnati, Ohio (closing): These cases were all tested after we thought they were well, so there is no question of suppuration.

In regard to x-ray exposure, we took that into consideration and tried to stay under the minimum. We figured it would take about 180 seconds to do any damage, so we stayed much below half of that. That is why we do not have these patients come back the next day, because we might be taking a chance as to what happens in the antrum. We get the function on that day and we are finished.

The reason we favor oil as against barium in spite of the fact that it is heavy, is that it has a therapeutic value and certainly will not do the patient any harm, whereas we are a little afraid of barium.

The suggestion that these cases be examined repeatedly is a good one. I have had that done myself. But it is hard to get patients to come in to have a study made because it takes four or five hours to do it. That accounts for the fact that we have not more cases. It is unfortunate that we have such a small number of cases, and the reason is that this work was all done in private practice and it is difficult to get the patients to come in.



## THE SELECTIVE TREATMENT OF MALIGNANCY ABOUT THE HEAD

By THOMAS G. GALLOWAY, M.D.

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The treatment of malignancy about the head has improved markedly within recent years. A great part of this progress has been in irradiation. Some of it has come from the employment of electrosurgery. Probably the most important factor, however, has been the better understanding of the relation of treatment of the tumor to its type, grade, location and other factors, and the more careful selection of the particular method for each case to give the best result. Though this must still be controversial, this discussion will attempt to define criteria for that selection of treatment.

The treatment of cancer is a many-sided problem and it should not be approached from any single viewpoint. The physician who is committed to any one invariable measure is likely to give his patient much less than his best chance of cure. Cancer requires for its best result the combined knowledge and experience of all the men who can give important information.

That includes the pathologist who has studied tumors from the point of view of their reaction to irradiation, their natural course and their prognosis, the radiation therapist, and a surgically trained clinician. The latter should be capable of any exposure or operative procedure in his field, he should know how to use electrosurgery to the best advantage, he should have studied the various methods statistically and in individual cases and he must have the ability to preserve the judicial attitude in the face of improving technic. Any treatment which does not take into account the full potentialities of both irradiation and surgery (usually electrosurgery) is likely to fall far short of its goal.

In addition it is necessary to have abundant quantities of radium or high voltage x-ray, or both, and modern diathermy equipment. Given such a team and such equipment, any cancer about the head and neck, seen reasonably early, should have a fair chance for cure.

Though we may dream of a time when some general agent shall eradicate every cancer cell from the body—and some recent work is encouraging—for the present irradiation and electrosurgery dominate the field. Each has its advantages and disadvantages and each its proper sphere, which has been fairly well defined by increasing

agreement among men who work with cancer. The division of the field will vary somewhat with the experience of the physician, with his ability to use either agent to its best advantage and with the type of his equipment or the quality and amount of irradiation available. No man lacking in either equipment or experience should proceed alone and the best results will be obtained in our field, I think, by a team of radiologist and laryngologist working with a competent pathologist.

The advantages of electrocoagulation, where it is applicable have been repeatedly stressed.<sup>1</sup> They include rapidity and ease of operation, relative lack of hemorrhage, of shock and of postoperative pain, the sealing off of blood and lymph spaces against infection and metastases and smooth healing. Tissue, malignant or benign, exposed to electrocoagulation is finally and irrevocably destroyed. It fails only because cancer cells are inaccessible to it or are in outlying strands or islands, or have already escaped to regional glands. It is destructive and necessarily sacrifices some sound tissue, usually a minimum of one centimeter about the growth. It has very definite operative risks, chiefly those of anesthesia, aspiration and hemorrhage, although these are far less than in scalpel surgery.

The advantages of irradiation are obvious. It does not terrify patients into avoiding treatment or temporizing. It usually leaves a good cosmetic result. It captures within its sphere of action outlying strands or early metastases that electrosurgery would miss. Its proper use is attended with little immediate risk.

Irradiation if improperly used has many bad effects. Insufficiently screened, it leads to destruction or very painful reactions of periostitis and perichondritis. Inadequately used, it may possibly stimulate cancer cells or seal them in scar tissue, hiding them until too late for surgery. It may, by action on the cancer bed, make the cancer radio-resistant so that proper treatment later is without avail. It may so impair nutrition that later x-ray, radium or surgery causes terrific destruction and usually if the first treatment does not succeed it fails—except as preoperative radiation may be followed by surgery at a short interval afterward.

It must be remembered that to be efficient either radium or x-ray must be used up to the maximum of normal tissue tolerance with a rather marked unpleasant or painful reaction, the so-called epithelite or epidermite reaction and with very definite and occasionally severe absorptive effects.<sup>2</sup> It affects most as a rule, the very tumors in which electrosurgery has fewest successes and these two agents, after all, make a very good team.

We cannot speak too dogmatically about radiation until we understand more specifically how it works. Even the recent report of the British Medical Research Council admits that the reaction to radiation is most complex and bewildering.

Regaud believes that action is on a susceptible nucleus; Ewing and Stewart think both nucleus and cytoplasm are equally affected. Other authors believe that a reactive fibrosis is the important factor and Pullinger believes that all effects of irradiation are due to vascular stimulation and vascular degeneration. There are probably two factors, early cancer cell necrosis and later destruction due to tissue reaction.

Very much has been written on radiosensitivity, and Stewart<sup>3</sup> required eighty-five pages to merely touch upon the high points of the subject.

He summarizes some of his conclusions as follows:

1. Sensitivity is only relative and ideas of tumor sensitivity will change with radiologic technic.

2. The effect of irradiation is a complex involving tumor cell, tissues of the host and possibly general reactions.

3. Radiosensitivity may mean rapid tumor regression, progressive regression over days to weeks or slow atrophy of months to years.

4. Radiosensitivity increases with the degree of embryonal type and usually with anaplasia, but not all anaplastic tumors are radiosensitive.

5. Tumors are more radiosensitive in young, less so in old or cachectic or anemic patients.

6. Certain tumors are inherently resistant—*e.g.*, melanomas and certain neurogenic tumors and desmoplastic tumors.

7. Normal tumor bed is favorable. Bone, fat and cartilage are unfavorable.

8. Infection interferes with irradiation.

9. Bulky tumors are resistant after infarction and liquefaction or with much secretion.

10. Metastases may vary from the parent, often are more resistant.

11. Rapid initial regression may lead to incomplete treatment, recurrence, radioresistance and failure.

The selection of proper treatment involves far more than histologic grading in given types, and the final judgment should be based on the gross appearance, clinical course, location and origin, previous treatment and local or general conditions of the host that help or hinder the best effect of radiation.

Yet the microscopic appearance, properly weighed, is of much importance. Definite types of tumor, as, for example, transitional cell carcinoma and so-called lympho-epithelioma, are very sensitive to radiation. Basal cell carcinomas are sensitive, as are round, reticulum-celled and lymphosarcomas. Adenocarcinomas and fibrosarcomas are relatively resistant.

Epidermoid carcinomas lend themselves to grading on the basis of relative maturity of cell types, abundance of stroma and other factors, and in Quick's<sup>4</sup> experience the gradings have been correct in the prediction of the expected action of radium within 10 percent error.

The degree of anaplasia is a rough indication of both malignancy and radiosensitivity, though lack of cell maturity, relative malignancy and radiosensitivity do not exactly parallel each other.

Melnick<sup>5</sup> and others have pointed out many possible errors in attempts at grading. Tumors of identical histologic appearance may run entirely different courses. Histologic criteria are not well established. Cell types are sometimes almost impossible to determine. Cells may be polymorphous. Mitotic figures may be inconstant, and there is growing evidence that amitosis or shortened form of mitosis may be important in tumor growth. In addition, as Melnick shows, a biopsy specimen may not be representative of the tumor as a whole; the character of the tumor may change with time, with blood supply, with irritation, infection, compression and other extraneous factors, and metastases may appear much different from the primary growth.

On the whole, however, histology may give valuable indications for treatment. Following the lead of Dr. R. Jaffe at Cook County Hospital, my clinical experience has been quite definite that cases with well matured cell forms, with much stroma, with hornification or other indication of specialization yield well to electrosurgery and often poorly to irradiation. Basal cell carcinoma, of course, gives good results with either. Very immature cell forms, undifferentiated, with little stroma, approaching grade IV of Broder's, yield much better to radiotherapy.

Location of tumors, in part at least due to the nature of the cells of origin, is an important factor in determining their treatment. The more radiosensitive tumors arise from the tonsillar regions, the pharynx and posterior third of the tongue. Favorable sites for electrosurgery are the skin of the face, ear, nose and lips, the maxillary sinuses, the palate, alveolar ridge, the cheek, the epiglottis and anterior third of the tongue.



The clinical appearance may be quite as important as other factors in determining treatment. If a fissured infiltrating tumor with poorly defined margins presents itself we can be doubtful about results with electrosurgery. If it involves in addition an unfavorable location, as the base of the tongue or lymphoid tissue of the pharynx, we may not waste our time and energy on it.

If it seems of short duration—though history is often to be doubted—and it seems to have grown rapidly, radiotherapy probably should be chosen. If there is much fixation and induration, except in long standing cases, it is probably not suitable for electrosurgery. If the tumor has a well marked or rolled border, or is papillary or fungating, it is likely to be amenable to diathermy.

If regional glands appear early or are disproportionate to the original growth, the tumor is more likely to be of immature type and radiosensitive. The dissection of regional gland areas is of doubtful value in my experience and may actually remove an important barrier except that discrete unfixed masses left after radiation may well be removed. Preliminary radiation of glands and later interstitial implantation after reflecting the skin seem to give better results, with much less effort to patient and surgeon. It must be remembered, however, that metastases are frequently more radio-resistant than the original growth.

We may say then if a tumor is of a relatively mature type, located in resistive tissue, of slow progress, of an appearance denoting relative limitation, the major reliance may be upon electrosurgery. If just the opposite obtains we should yield it willingly to irradiation and leave our energies for more likely material to the great improvement of our personal morale and of our statistics.

Preoperative x-ray or radium may be very valuable, both to the regional gland area and to the local lesion. It may also be valuable as a therapeutic test to determine the radiosensitivity. It may reduce the bulk of the tumor and its troublesome vascularity, as in the case of a hemangioma, a nasopharyngeal fibroma or chondrosarcoma. If it fails to completely eradicate the tumor, electrocoagulation can then finish the job.

Postoperative irradiation gives additional protection and should be done when there is any question of gland involvement or complete removal. If used it should be thorough.

General measures of importance include correction of asthenia and anemia, good nutrition, especially through high vitamin diet and good mouth hygiene. We have recently seen a case with multiple skin

carcinomas of three histologic types running wild brought under control by electrosurgery and high vitamin A diet.

The treatment of special regions may be briefly considered. On the skin, basal cell carcinomas yield well to irradiation or coagulation or desiccation with about 95 percent cures or more if early. On the lids radium is probably better because it does not scar. Squamous celled cancers, I believe, are better coagulated, though irradiation, usually in caustic dose, will give 75 percent cures in cases not too old. Combined treatment is excellent. For the lip, Pfahler's<sup>6</sup> statistics for electrosurgery followed by intensive fractional irradiation seem as good as any.

Carcinoma of the antrum, even of an immature cell type if still confined to the bony cavity, yields well to electrocoagulation, but exposure must be adequate and destruction radical. Preoperative and regional gland radiation and radium may be used in the cavity but must be very well screened to prevent painful periostitis.

If carcinoma of the tongue involves the base or is fissured or deeply ulcerating, radiation is the treatment of choice. If early, papillary and involving the anterior third or border, electrocoagulation will give good results, especially if combined with radiation. Electrosurgical removal of the whole tongue is safe, easy and not too mutilating, if radium fails locally, with glands uninvolved or controlled.

Carcinoma of the cheek will respond well to either endothermy or radium. If it involves the alveolar ridge or maxilla or mandible, coagulation, I think, should be used.

I have one five-year cure of carcinoma of the tonsil by coagulation among numerous attempts. On the other hand, Duffy could report 20 percent and Coutard 28 percent of five-year cures in this region by irradiation, and these cases should surely be given over to x-ray or radium.

Carcinoma of the epiglottis and occasionally of the velum may be completely removed by electrocoagulation, but all others of the nasopharynx, pharynx and hypopharynx should be left to radium and x-ray. There is little justification, I think, at present for the formidable approaches to these tumors by an external route unless, as rarely happens, they are of a very mature cell type, and even then suspension and coagulation will usually give adequate exposure if they are operable.

The radiotherapists have discovered that x-ray or radium, well screened, using different ports of entry, may be used with relative safety in the presence of cartilage. With much enthusiasm, though the case does not yet seem statistically proven, some of them have

seized upon carcinoma of the larynx as their own. The best figures for irradiation appear to be those of Coutard,<sup>2</sup> showing about 26 percent of five-year cures, although the seventeen cases of 1926, reported in 1931, gave 53 percent cures, quite possibly showing a marked improvement in the technic. On the other hand, Jackson reports 82 percent and Gluck-Sorenson 88 percent of five-year cures by laryngofissure in selected cases. St. Clair Thomson has pointed out that laryngofissure leaves a nearly normal individual with natural airway and serviceable voice. We should, I think, hesitate to turn such early cases to any more problematic method until time has proven its value.

Laryngectomy, I think, is still in more advanced intrinsic cases, with its 76 percent cures given by MacKenty, to be preferred to radiotherapy, although it is easily possible that the latter may yet prove to be the method of choice. Radiation may give additional security as an adjunct postoperatively and is the choice, I think, in nearly all extrinsic cases. Coagulation is at times of value in cleaning up doubtful borders after laryngofissure and may at times be better than laryngectomy, if used through a thyrotomy wound after cartilage resection in cases which have overflowed the larynx above. The methods of Ledoux and Harmer or the method of Hautant and Coutard with thyrotomy and mass destruction of the tumor, though incomplete, followed by irradiation or implantation, seems of much value, as shown by three cures in Coutard's statistics of cases of 1926.

Whether x-ray or radium is better is still debated by those specializing in their use. Hard gamma rays reaching the depth of the tumor are the effective agent and can apparently be secured by either with proper filtration. Berven feels that the radium bomb is much superior, but Regaud believes both about equally effective. X-ray seems definitely cheaper. Radium is necessary for localized or interstitial use. To one who has observed their effect it appears that the use of either is dangerous if insufficient, because it makes tumors radioresistant, and if radium is used it must be well screened and used up to the point of maximum tolerance. If x-ray is employed it must be of high voltage—probably not under 200 kilovolts, given in fractional doses after the method of Coutard—well filtered, also to the point of greatest skin tolerance. Certainly there is no place for the careless, the casual, the inadequate use of either x-ray or radium.

This discussion has attempted to show that the otolaryngologist should not be committed to any one invariable treatment of malignancy. It has tried to define the best indications for the use of both

irradiation and electrosurgery evolved by the cooperation of radiation therapist, pathologist and laryngologist.

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### DISCUSSION

HARRY L. POLLOCK, M.D., Chicago, Ill.: As this paper is confined principally to therapeutics, let it be understood that the best end results can be obtained only by a close, cooperative team consisting of a pathologist, an expert radiologist (and by that I mean, one understanding fully the action of the different rays in all their varying strength and potentialities as well as having had a wide practical, clinical experience) and lastly an otolaryngologist who is at all times in direct charge of the patient.

X-rays and radium inadequately employed have only a detrimental effect on the recovery or amelioration of the condition. Is it, therefore, to be wondered at that unfavorable results invariably occurred in earlier years when the laryngologist with his limited knowledge of this new field, employed radium in small doses?

At the present time there is still much discussion as to whether surgery or radiation is to be employed, whether alone or in conjunction with one another. Shall the rays be used first and then surgery, rays alone, surgery alone, or surgery followed by rays?

I sincerely believe that with our present knowledge of radio-sensitivity, pathological classification of the tumor as well as its location and duration, we are arriving at a better understanding as to which method is the most efficacious in that particular tumor. Where surgery is indicated and to be employed, I firmly agree with Doctor Galloway that electrocoagulation is far superior to the cold scalpel for the reasons mentioned in his paper. During the past few years we have learned a great deal regarding the tumors which are radiosensitive or radioresistant, both from a pathological as well as clinical standpoint. For example, we know that transitional cell carcinoma and lympho-



epithelioma are highly sensitive to the gamma rays; on the other hand the squamous cell and adenocarcinomata are very resistant. We recognize that the former are very prone to metastasize early and the latter, quite late. I have seen these radiosensitive tumors disappear readily under proper x-ray or radium treatment but unfortunately the metastases which occur very early are much more radioresistant than the original tumor. Tumors of this type are usually found in the tonsillar area, base of tongue and pharynx and, in my opinion, should only be treated by rays unless a small portion remains which should be removed by the electro-knife and then again rayed.

I differ with Doctor Galloway regarding the early lesions of the lip, face and anterior portion of the tongue because if the lesion is accessible, and it usually is, implantation with radon seeds or application of radium plaque gives most excellent results.

Notwithstanding the excellent results which Trotter of England has reported on block resection of the cervical glands, I am still of the opinion that they can be better cared for by use of rays. If only the submaxillary gland is involved complete resection should be carried out and followed by x-ray treatment. Carcinoma of the antrum should always be attacked surgically and very radically, followed by x-ray or radium.

Regarding carcinoma of the larynx, I believe there is a greater divergence of opinion relative to its treatment. In early intrinsic cases I am certain that removal of the growth by electrocoagulation through a laryngofissure offers the best results. Jackson has 82 percent cures in early cases, cures ranging from five to thirty-one years' duration. I am sure that x-ray and radium cures would fall far below that figure. When there are metastases in these intrinsic cases I believe that surgery followed by intensive ray therapy will give the best results. In extrinsic carcinoma of the larynx I should put my faith in the ray treatment alone.

Judging from the combined experience of many authorities, we are agreed that early diagnosis is the essential factor to a good prognosis. In Jackson's cases of carcinoma of the larynx, 19.3 percent were classified as operable and 81 percent as inoperable. Is this not a stigma against our profession? I sincerely believe that if propaganda on the subject of early diagnosis were increased and more particularly directed to the attention of the general practitioners who see most of these cases first, we would be afforded an opportunity of examining these patients earlier, thereby averting much suffering and restoring many individuals to normalcy.

HAROLD M. TRUSLER, M.D., Indianapolis, Ind.: Concerning the use of x-ray and radium, I prefer to consider them as valuable adjuncts to surgery or electro-surgery. Though it is true no doubt that radiation alone may sometimes cure skin cancer, it fails or is improperly used very often, and we surely must agree that these lesions—when early and accessible—should be excised preferably by electrosurgery.

This is especially true where the lesion closely overlies bone or cartilage. In my experience a skin cancer, which has ulcerated into the bone or cartilage, does not respond well to x-ray and radium.

A delay caused by giving radiation a trial may seriously increase the mutilation.

EDWARD KING, M.D., Cincinnati, Ohio: Recently I saw a patient who had carcinoma of the left cord involving the left arytenoid. He was advised to have a laryngectomy. Doctor Cutler of Chicago had been in Cincinnati a couple of months previously, and while I did not hear him myself the family doctor had heard him and was impressed with the results he was getting in carcinoma of the larynx, so the family doctor persuaded this man to go to see Doctor Cutler. After treatment by Doctor Cutler this man came home with a normal larynx. Since then I have seen him twice—he has been under my observation for about four months following that treatment, and he has a perfectly normal larynx. He is a salesman, is married and has eight children, so in that case radiotherapy was certainly preferable because laryngectomy would have made him a charge on the community.

I can readily see from Doctor Galloway's paper that we can be driven from our sound position, as the results from surgery of the larynx to referring these cases for radiotherapy instead of proper surgical therapy, unless we have such men as Doctor Galloway to guide us.

JOSEPH C. BECK, M.D., Chicago, Ill.: I want to mention just one point apropos of what Doctor King has said, except that I go farther. Doctor Galloway brought up the question of very early carcinoma of the larynx, meaning even those that are operable by laryngofissure, but it takes a great deal of courage for a laryngologist to submit a case like that for irradiation, even by the best experts. Yet how are we going to be absolutely fair in regard to this matter unless we do that—submit the squamous cell carcinomata in the middle portion of the cord, so small that you could almost remove it by the direct method, to irradiation.

I am pleased to say that in the Michael Reese Hospital in Chicago Doctor Cutler is in charge of the tumor clinic, and I have, as laryngologist, turned over two cases, and those cases are being very carefully watched as to what will result, not in three months, six months, or a year, but really in the period of time in which we know recurrence may occur after these growths have disappeared. The growths, which were shown to be squamous cell carcinomata, were removed early, and were in well, strong individuals, otherwise healthy except for these growths; they were both submitted to irradiation, x-ray and radium. They developed the secondary changes that come from such treatment, externally and internally—changes in the epithelium; they have healed, and now these patients are being watched as to changes in the tissues. If these cases remain well I think we can say "Amen" to that type of treatment and shall have a right to speak about the curability or inefficiency of the treatment. I still maintain that there are cases that must be submitted to that type of treatment, but the greater number of cases, both of laryngofissure and laryngectomy, will continue to be operated upon because we know what we can do with that kind of treatment.

THOMAS G. GALLOWAY, M.D. (closing): Preoperative radiation was mentioned. It cannot be extensive or very intensive. If there are glands involved I do not see how you obtain beneficial radiotherapy. You cannot discover the glands soon enough. That is one argument against preoperative radiation.

Stimulation by x-ray really does have an effect upon the tumor bed. Some tumors grow faster after x-ray treatment.

Measuring the voltage—Coutard's first cases reported were done with a 175-voltage pressure, and as we go above 200 kilovolts the hazard is increased slightly, but there is a question whether it is necessary to go to 700 to 1,000 kilovolts.

Doctor Pollock, of course, with his clinical experience, is right in most things. I am at the County Hospital where we see the end results of the failures in Chicago. Patients come to us as a final haven, when they have had unsuccessful treatment and have spent all their money in radium institutes. We have seen many cases that showed beautiful results and then three and a half years afterwards recurred. Coutard's statistics show that they did get results for five years, but a good many die between seven and eleven years. We have one case in which there is recurrence after twenty-three years of radiation. Of course, that patient has been symptom free in that time, and that is an argument in favor of radium, but we cannot talk about radium cures for a shorter period than five years.

You can get beautiful immediate results with a very inadequate dose of x-ray. I recently saw a case that had been promised a cure by a certain well-known clinic in Chicago, and he came in to die. He died of bronchopneumonia after a tracheotomy. He was a very sick man and did not want to live. He told us he had been promised a cure. Another patient recently came from the same radiation clinic, with the impression that he had been promised a 100 percent cure. He had the smallest tumor I have ever seen at the junction of the anterior and middle third of the cord, something that could almost be done by suspension.

I think Doctor Beck is right—irradiation can cure those cases much better than those of an infiltrating character. But it seems a shame to refer such cases to radium when we think of the results from laryngofissure by the St. Clair Thomson technic.

Doctor Trusler and I agree. This question of radiation is improving and we all have had our viewpoint shifted. We should preserve an open mind. I think we must be strong for irradiation and give the radiologist every chance. Maybe we will never operate on carcinoma of the larynx.

## PATHOLOGY OF OTOSCLEROSIS

By E. W. HAGENS, M.D.

Chicago, Ill.

In considering the pathology of otosclerosis one finds in the literature<sup>1</sup> a large number of reports and considerable differences of opinion as to the interpretation of the findings. Most of the discussions have centered about the changes in the bony capsule of the labyrinth. However, Gray<sup>2</sup> has written several times, and again lately, about changes in the eighth nerve. He believes he has found a localized loss in the neurokeratin of the medullary sheaths and a similar absence in the neurilemma of the cochlear nerve. These changes were absent in the vestibular division. Other observers have found degeneration in the organ of Corti, the spiral ganglion and the eighth nerve.

In considering these changes one must remember the normal difference between the two divisions of the eighth nerve. The fibers of the vestibular branch are normally larger, more distinct and more easily seen. The neurokeratin is visible, whereas in the cochlear division it is not made out. The neurilemma is normally seen in both branches. In the cochlear portion one notes that the fibers of the endoneurium are more abundant. A study of the five otosclerotic bones in this present report does not reveal any of the pathologic changes in the eighth nerve as described by Gray and others. The neurilemma was as definite in the cochlear division as in the vestibular. Thus it may still be questioned as to whether changes occur in the eighth nerve characteristic of otosclerosis. Degenerations in the organ of Corti and spiral ganglion have been found in cases with otosclerosis, but the degenerations would not necessarily have to be on the same basis as the otosclerotic bony foci. It would seem that to prove reasonably the connection between pathology in the several parts of the ear there should be a considerable number of cases showing similar findings; control cases should not show these alterations.

Returning to the consideration of the changes in the bone, one finds otosclerosis recorded in a child as young as one year and recently Guggenheim<sup>3</sup> has found what he believes to be an early stage of it in a seven-month fetus. Some of the theories as to the development of the otosclerotic bone are as follows:



1. Vascular constriction (O. Mayer).
2. Altered vascularization due to an inflammatory process in the tympanic cavity (Siebenmann).
3. Chronic local infection in the mucoperiosteum of the tympanum, such as following otitis media (Fraser).
4. Chronic inflammation (Manasse).
5. Venous stasis (Wittmaack).
6. A gradually increasing defect in the vasomotor mechanism which governs the nutrition of the ear (Gray).
7. Development from the part of the labyrinth last ossified (Pierce).
8. Regression (Guggenheim).

Practically all observers agree that there is probably an hereditary factor in these cases and that the condition is usually bilateral. The various general factors which may influence the condition are syphilis, tuberculosis, gastrointestinal toxemia, toxemia from any focus of infection, alterations in the function of the glands of internal secretion, faulty diet (lacking in vitamins), arteriosclerosis, pregnancy.

The change in the bony labyrinth in otosclerosis is characterized by occurring in localized patches, termed foci. No other bone disease is exactly like it, although several are similar to it except for the focus formation. The commonest site for a focus is just anterior to the foot-plate of the stapes. This has been called the "site of predilection" and it is here that the fissula ante fenestram is found. Other foci are found, however, (a) surrounding one side of the cochlea and often extending to the internal auditory meatus; (b) about the round window; and (c) about the semicircular canals. The focus of otosclerosis is usually found to consist of a spongy type of bone with vascular marrow-like cavities. The majority of cells about the vessels in these spaces are probably osteogenetic in type. The bone cells are very irregularly laid down, haversian systems either being absent or infrequent. Osteoblasts and osteoclasts are frequently seen in the focus; in some cases they are very scarce, in other cases more plentiful. While some observers have found these cells at the focus border adjacent to the normal bone, others have not seen them in this location. At the "site of predilection" the focus frequently develops towards the stapelial foot-plate and may invade and replace more or less of it. It may even form an exostosis towards the cavum tympani. Vessels from the focus often connect with the overlying mucous membrane of the cavum tympani. The subepithelial tissue may be quite vascular or it may be thickened and fibrous. In many foci finger-like projections, usually surrounding a vessel, ex-

tend out from the focus into the surrounding bone. The foci appear to develop at the junction of the enchondral layer of the bony labyrinth and the periosteal layer. As the focus develops, the periosteal, enchondral and endosteal layers become involved. The focus takes a striking bluish-red stain with hematoxylin-eosin which can often be recognized without the microscope. The cartilage remnants so often seen normally in the bony labyrinth are not found in the focus.

Two main views have been held as to the enlargement of the foci. The first view postulates that the normal bone is absorbed by osteoclasts and this is followed by the otosclerotic bone. The second view is that the focus is a tumor-like formation and that it destroys the normal bone as it develops. The frequent lack of osteoclasts at the edge of the focus speaks against the first view, and the absence of a common tumor cell argues against the second view. It is possible that the bone adjacent to the focus is prepared by chemical action and that the focus enlarges by sending out the finger-like processes into the chemically altered bone, gradually replacing it. Some investigators have held that the otosclerotic process is inflammatory, while others, failing to find round cells or lymphoid cells, have denied this. Otosclerosis may be found alone, or it may be associated with other conditions such as acute or chronic otitis media, congenital malformation, cretinism, osteitis fibrosa, neurofibroma of the eighth nerve, fragile bones and blue sclera, tuberculosis, leukemia, and Gaucher's disease.

I wish to present briefly five temporal bones showing otosclerosis from four patients. Four of the bones have previously been reported<sup>4</sup> in detail. Of the four patients, two were men and two women. The youngest patient was one of the women who died at the age of twenty; the oldest was one of the men who was seventy years at the time of death. This last case was the only one diagnosed as otosclerosis during life. The histories and findings in these cases are as follows:

*Case 1, Bone 1.*—Ta, a woman aged thirty-seven, died from intracranial complications following an acute suppurative otitis media. As far as could be ascertained there had been no previous history of deafness or ear trouble. Only the right or involved ear was furnished for microscopic examination. Study of this bone showed infection of the pneumatic spaces and, in addition, a definite focus at the "site of predilection" just anterior to the foot-plate of the stapes. The latter was not involved, but the focus did replace the vestibular lip of the joint. The fissula ante fenestram was absent except for its notch-like beginning at the vestibule. From the anterior joint of the stapes the connective tissue was seen to extend around the tympanic border of the focus in a fibrous-like layer. This was non-vascular and apparently non-inflammatory. The focus

bone was vascular, but showed few osteoblasts and practically no osteoclasts. In this case otosclerosis and acute suppurative otitis media were both present.

*Case 2, Bones 2 and 3.*—Fe, a woman aged twenty, died from otitic intracranial complications. Her history was that at the age of five she had had scarlet fever which had left her with a chronically discharging left ear associated with moderate deafness. No deafness was noted in the right ear. An acute exacerbation of her ear condition led to fatal complications.

Examination of the left ear showed considerable infection but, in addition, several foci of otosclerosis. The foci were found at the "site of predilection," about the cochlea extending from the apex around to the internal auditory meatus, and about the posterior semicircular canal. The first mentioned focus had invaded the stapes and replaced nearly all of its foot-plate. In addition, the bone was much enlarged, forming a hyperostosis. A distorted anterior joint ligament could be seen and connective tissue from this extended around the tympanic border of the focus as a fibrous, non-vascular, non-inflammatory layer. The fissula ante fenestram was not seen. Osteoblasts with new bone underlying, and osteoclasts were seen in the focus which was quite vascular. This bone revealed three foci of otosclerosis. Thus chronic suppurative otitis media and otosclerosis occurred together. Clinically, the deafness in this ear would be sufficiently explained by the chronic suppurative otitis media, the otosclerosis being entirely masked by the otitis.

The right ear revealed a focus at the "site of predilection," one extending around the cochlea as on the left side, one about the round window, and a fourth about the medial crus of the horizontal semicircular canal. The focus in the stapes region did not actually invade this ossicle, but did, however, form a definite hyperostosis about it. The appearance of the focus was similar to that on the left side. The mucous membrane overlying the focus on its tympanic side was thickened by dense, fibrous tissue. The fissula ante fenestram was not seen. In this bone there were four foci and yet the history did not reveal ear infection or deafness on this side.

*Case 3, Bone 4.*—W, a man aged seventy, died from organic heart disease. Doctor Shambaugh diagnosed otosclerosis seven years before death. There was a history of severe bilateral deafness and tinnitus for a number of years. Both temporal bones were obtained, but the right one was so altered during the preparation that only the left one could be studied accurately.

Examination of the left temporal bone showed single focus at the "site of predilection." The focus involved a considerable part of the stapedial foot-plate and anterior crus, producing a hyperostosis. An irregular connective tissue band simulated the anterior joint and continued over the tympanic surface of the focus. In places blood vessels were seen in the connective tissue. The bone of the focus was quite sclerotic, the marrow-like vascular spaces being quite small. Osteoblasts and osteoclasts were not seen. No fissula ante fenestram could be recognized. This case represents the older, more sclerotic type of focus.

*Case 4, Bone 5.*—Mi, a man, aged thirty-five, died with luetic meningitis. A gumma was found in the right cerebral hemisphere and the brain showed marked edema. There was no history of deafness or ear trouble except frequent otalgia with tinnitus as a child. The family history was negative as to deafness. Severe headaches, with pain back of the eyes, had been present since childhood.

Examination of the temporal bones showed an absence of suppuration on each side. The right temporal bone revealed no otosclerosis and the tympanic mucous membrane overlying the stapes and surrounding bone was normal. The fissula ante fenestram was easily identified. The left temporal bone revealed a focus at the "site of predilection." The stapes and its foot-plate was not involved. The focus was quite vascular; in one place a large finger-like vessel extended into the adjacent bone in the direction of the processus cochleariformis. Only a few osteoblasts and osteoclasts were seen. The fissula ante fenestram was indicated only a small notch at the vestibule. Of considerable interest was the tympanic mucous membrane overlying the focus and to a lesser extent about the crura. Examination revealed a greatly thickened subepithelial layer which was extremely vascular. Larger vessels with thin walls almost filled this layer. No definite evidence of past or present inflammation could be seen. This, then, represents the pathological basis for the clinical Schwartz's sign. Unfortunately, no record of the appearance of the drum membranes was made on the chart.

The interest in this case lies in the finding of a unilateral otosclerosis with a definite opportunity for comparison between the two sides. There was no history of suppurative otitis media. If otalgia meant a lesser type of involvement and if both ears suffered, as might be expected, yet otosclerosis developed only on one side and on this side the tympanic mucosa was extremely vascular, while on the other it was normal.

A summary of the findings in this series of bones reveals that most of the points made in the literature are demonstrated. All the foci stained prominently with hematoxylineosin. In a previous article<sup>5</sup> I have attempted to show that this may be due either to the unusual attraction of the focus bone for the mordant in the stain, or the presence of iron in it. A number of observers have thought the peculiar staining was due to the excess of calcium in the focus, but this could not be proved. In regard to the fissula ante fenestram, the foci were all too far advanced to tell whether they had started from this structure or had engulfed it in their development. The close connection of the overlying thickened tissue on the tympanic side of the focus would lead one to believe that the thickened membrane, vascular or not, as the case may be, was related to the focus and not associated with otitis media. This belief is further strengthened by the findings in the unilateral case, Mi, and by the presence of foci far from the cavum tympani as in case Fe. Also in case Fe the non-suppurative ear had more otosclerotic involvement than the suppurative one did.

Finally, one may say that no theory of etiology seems adequate at present. No descriptive term of the pathology is wholly satis-



factory, and the name "otosclerosis," while unsatisfactory, is still the best known word for the process.

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### DISCUSSION

ROBERT SONNENSCHNEIN, M.D., Chicago, Ill.: Just as in the discussion of the treatment of certain diseases, the recital of a long list of remedies means that none is satisfactory, otherwise only one or two would be mentioned, so the presentation of the eight theories regarding the development of otosclerosis demonstrates how diverse are the opinions and how lacking is our knowledge of actual facts regarding the development of this dreaded aural affliction.

Albert A. Gray's theory was presented in detail in May, 1934. It is based on the thesis that the "essential causative factor of otosclerosis is a gradually increasing defect in the vasomotor mechanism which governs the nutrition of the structures of the organ of hearing as a whole." In a word, Gray believes that one process involves the whole hearing mechanism. He mentions, as Doctor Hagens has stated, "a localized loss in the neurokeratin of the medullary sheaths and an absence in the neurillema of the cochlear nerve. The other theories of the causation of otosclerosis have been often discussed, but I believe it will not be amiss for me to quote a few details from Gray's Dalby Memorial Lecture (May 4, 1934):

There is no evidence whatever of any difficulty in any glands or their secretion in otosclerosis; neither is there any evidence in the bone metabolism of the body. On the contrary, the subjects of otosclerosis are, apart from their deafness, perfectly normal individuals with ordinary average health. The deafness of otosclerosis bears very little relationship to the extent of the disease in the bone. Deafness may be very severe when the stapes is hardly fixed at all. The severity of the tinnitus bears no relationship at all to the extent of the disease in the bone. The extent of the change in the bone bears very little relationship to the duration of the disease. The deafness of otosclerosis is to a large extent functional, and is a result of the insufficient supply of blood to all the nerve structures concerned in the perception of sound. The preponderance of women as subjects of otosclerosis is the result of greater instability of their vasomotor system, and the more frequent disturbances to which it is exposed. The changes in the bone show a remarkable bilateral symmetry, even to minute details. This symmetrical distribution is readily explained by the writer's view of the causative factor of otosclerosis. The vasomotor nerves governing the nutrition of the organ of hearing are anatomically symmetrical like other nerve structures in the body. If, therefore, structural changes occur as a result of defective functioning of those nerves, such structural changes will, naturally, be bilaterally symmetrical in their distribution.

This theory certainly sounds quite plausible, and especially so since the causative factor, if it is the true one, would account for many of the charac-

teristics of the disease. However, Doctor Hagen's specimens do not show these findings. In a word, we cannot say that this theory is the one which explains every phenomenon satisfactorily.

Although it is not important so far as this paper is concerned, I would like to state that it has always seemed to me that Shambaugh's term "otospongiosis," more clearly describes the pathology than does "otosclerosis," but the latter term has become so fixed in the literature that even if considered inadequate, it could not be eliminated.

Mere speculation or theorizing is unproductive of results in a problem like that of otosclerosis. Extensive research work from every angle, and careful histologic work like Doctor Hagen's, together with postmortem examination of temporal bones, study of clinical cases and animal experimentation, will need to be continued in order to solve this very puzzling problem.

H. G. LAREAU, M.D., Chicago, Ill.: All the theories mentioned have been based on histologic findings. I want to call attention to another method of approach that has come into use in the last few years, which we may call the biochemical approach. None of these investigations have led to anything that might be dignified by the name of "theory," but they are something that might stimulate thought.

Some years ago before this society, and at Washington last year at the American Otological Society, I presented a few specimens showing the effect of chlorin treatment. The biochemical approach shows that the nutrition of the bones of the labyrinth is influenced by chemicals, which may help in the management of some of these cases.

FRANK J. NOVAK, JR., M.D., Chicago, Ill.: I would like to discuss two points in this paper. First, the case in which there are large blood vessels. I would like to ask what is the interpretation of this situation. We have a considerable number of blood vessels of wide caliber and thin walls; they are not capillaries but blood vessels—really they are blood sinusoids.

Second, as to Case 4, in which we see this layer of osteoblasts and this layer alongside of it. I would like to ask Doctor Hagen what he observed in this tissue under oil immersion, whether it had a more or less granular appearance, like dust. I observed in the turbinate of an individual with progressive deafness a similar thing, and when the turbinate was removed we found a layer of osteoblasts and alongside of it a layer which stained entirely differently from any other portion of the bone. It has a light, sky-blue tint and is peppered with very fine granulations which are observable only under oil immersion. The rest of that turbinate showed definite changes of an osteomyelitic process—lines showing absence of calcium. I would like Doctor Hagens to inform me whether he has observed the same thing in regard to color and granulation.

M. L. MCCARTHY, M.D., Cincinnati, Ohio: Doctor Hagens has presented for us a clear and concise report of his findings in this group of cases in which sectional studies of the temporal bone have been made. His answer to Gray's assertion that changes in the cochlear division of the eighth nerve were found in the group of otosclerosis cases which he studied, is that his findings and those of Gray are not the same. He does not say that such changes are not possible, but states with commendable restraint that in his group of cases there were no findings to suggest degeneration of the cochlear division of the eighth nerve.

In discussing Doctor Hagen's paper, it is fitting to touch briefly upon the difficulties attending the setting up of an organization which permits such careful studies as he has here presented. The labor attendant upon the selection of cases, the supervision and preparation of the preliminary records (and I note with some degree of comfort, that one of his base histories was lost, an experience which has surely fallen to the lot of most of us in the preparation of a paper) the careful removal of the temporal bone as soon as possible after death, the problem of decalcification and the painstaking supervision of the sectioning, staining and mounting of the specimens are often passed over too lightly.

The tremendous consumption of time before even one has opportunity to study the sections, and then the prolonged labor over finished sections, can be easily misleading to the worker himself. In even the most efficient centers of education, with ample funds for skilled assistance, if the worker is able to prepare, section, study and classify a dozen temporal bone preparations in a year he can feel that he is doing very well. It is not to be forgotten that the dozen preparations in turn represent only six cases. If any worker in malignancy were to report on twelve preparations from six patients, and then attempt to draw conclusions as to the etiology of carcinoma, one would be rightfully skeptical as to the value of such deductions. A vast number of malignancies have been sectioned and studied for over fifty years, and we are just now beginning to approach the acceptance of a conclusion as to the cause, so that we should be most patient with the worker in otosclerosis who, as Doctor Hagens has done, carefully records his findings and draws no hard and fast conclusions. The tendency is to regard the time and effort spent as a substitute for the number of cases examined. In this light, a review of the early years of the study of cancer is very illuminating.

I should like to ask Doctor Hagens briefly to evaluate the work of Weber in his use of polarized light for the study of his sections, and that of Fowler, who has used ultra-violet light for the same purpose. I should also like to ask if in normal preparations he is always able to recognize the fissula ante fenestram.

May I also ask Doctor Hagens whether he agrees with the statement of Moritz Weber, who says that shell bone resulting from the epitheloid arrangement of the osteoblasts does not occur in his sectional studies of otosclerosis. The answer hinges on whether these osteoblasts are in epitheloid arrangement. I notice also that the author, while using hemotoxylineosin stain, avoids the term "blue, red or white mantels," as first described by Manasse and later elaborated upon by Moritz Weber who used the same type of stain. May I ask whether he agrees with this statement of Weber that otosclerosis is not a disease of a purely primary focus, but of the entire labyrinthine capsule.

Finally, in addition to the fine work which has been done in this country by Doctor Hagens, there is the illuminating and painstaking work of Moritz Weber, the Fowlers, Guild and his co-workers at Johns Hopkins, Guggenheim, and others. With the inclusion of Bast (whose monumental work on the osteogenesis of the human periotic capsule is an example to all research workers in this field), in the previously mentioned group, we come to realize that as fine as the effort to date has been, it has been almost exclusively along cytological and embryological lines. There is no question that the ultimate of cytological detail must be worked out, and all such efforts help tremendously to guide us to the solution of the problem. However, we shall probably be long delayed in a com-



prehension of this situation, unless the cytologist, the clinician and the physicist work together.

Unless signs fail, we are about to enter another phase of the endeavor to solve this engaging and baffling problem. Presently we shall have an answer as to whether there are synchronous changes taking place in the other bones of the body resembling otosclerosis. There will be clarification of the relationship between osteodystrophia fibrosa (Paget's disease), osteogenesis, imperfecta, and otosclerosis.

The experimental work which has been done to date is pitifully inadequate. Entailing as it does laborious cytological check-ups, the experimental approach to this problem presents enormous difficulties. In spite of this I have no doubt at all that in time these difficulties will be surmounted. Until then each portion added to the sum of our knowledge is important. Doctor Hagens has ably and concisely stated his findings and his conservative conclusion that no theory of etiology seems adequate at present, is to be commended.

E. W. HAGENS, M.D. (closing): Taking up Doctor McCarthy's discussion, I have had no personal experience with polarized light or ultraviolet. I am interested, and if I were able would study my sections with that in view; but I cannot speak of the results that are reported.

In regard to observing the fenestra, I think we can find fistulous fenestra in most temporal bones if we section and look for it. In some of my sections made early I did not demonstrate it, but I think that was my fault. Certainly in later years when I attempted to find it I was able to do so. It varies, however, considerably, as brought out by Doctor Wilson. I recently heard Doctor Wilson's paper where he showed models and slides. The fistula apparently connects the vestibule with the anterior crus and is the channel that runs to the tympanic surface. It has been found filled with connective tissue and with vessels. It is also surrounded with cartilage in a number of cases and in some may be found a large focus of cartilage remaining at that site. I recently sectioned a three-year old who has large focus of cartilage at that point and connection with the fistula. Bast states he does not know the connection nor can he describe it, as to whether the focus of otosclerosis starts in the fistula or not. In one of his specimens Bast states he thinks the focus started about the fistula proper, but in mine the foci were so far advanced that it was obliterated.

In regard to osteoblasts in the new bone underneath, I believe these are osteoblasts or bone cells which usually take a pink stain. In regard to staining, it is difficult to go back in the literature and tell what they are talking about because the stains vary. You may stain one way, or another, and they will differ in appearance. It is difficult to decide when one man reports a blue tint and another yellow; it depends on hematoxylin. I did look at it under oil immersion, but I do not recall any stippling or dust formation, but I will look at sections again and report to Doctor Novak in Chicago.

In regard to the blue mantels, I failed to mention them for lack of time. I mentioned some of them that I was interested in, and others I have spoken or written about before. My sections show them but I think it may be the way the process progressed, sending out finger-like vascular processes into the bone and then these blue mantle layers form around them.

As to whether otosclerosis involves the whole bony labyrinth, certainly the focus by which we make a diagnosis is localized. I cannot believe otosclerosis



is a general thing. It may be, but it cannot be proved because the thing by which we diagnose it is the focus formation. That brings up Wilson and the question Doctor Mullin asked. In Doctor Wilson's specimens he found the whole bony labyrinth involved, but there is a difference between the anatomy of the chicken and the human. So while it is similar to otosclerosis, and under low power might look identical, the difference is that in the chicken it is generalized and in the human it is localized.

As to whether the other bones show sclerosis, that was mentioned by one or two men in the literature, but whether they were quoting someone, I do not know. I do not think it has been found outside of the petrous bone. One man reported it in the ossicles. I think there may be a reason for that. I do not think there is another bone in the body like the petrous bone in its development, and it may be because of that peculiar development that it forms a focus. I do think we should look for otosclerosis in other bones, but these cases come in without diagnosis and you find it on routine examination. There should be more routine work done.

Doctor Mullin asked whether I was talking about pathological otosclerosis or clinical. I am talking about pathological otosclerosis. I believe they are different. I believe the foci are about the stapes. I think clinical otosclerosis is much more uncertain than pathological. If we did more routine work we would find out whether the clinical diagnosis is correct. We think we are right, but we should check them up and we would likely find we had missed a good many clinical cases which had not involved the structures that would give rise to deafness.

I was glad Doctor LaReau brought out the point he did. I have read of this biochemical approach, but did not mention it because it did not fit in with the type of paper I was presenting.

As to the blood vessels that Doctor Novak mentioned in the last case. You cannot call them arteries, you cannot call them veins; they are fairly close to the sinusoid type. In studying the sections one of the men at the university thought they were of the type that show the stasis type of stain, and not the inflammatory type.

## SOME CONCLUSIVE REMARKS REGARDING PLASTIC SURGERY FROM PERSONAL EXPERIENCE

By JOSEPH C. BECK, M.D.

Chicago, Ill.

A recent visit to this country by Sir Harold D. Gillies, of London, brought him to Chicago, where he delivered the annual Charles Mayo Northwestern University lecture.

He chose the subject of "Plastic Surgery," and the writer had the pleasure of hearing him. It was what he said regarding the possible future of plastic surgery and the duty of the surgeon to the public that prompts me to bring this subject before you for your consideration. Gillies is considered one of the outstanding contemporaries in this work, and having watched him from the time I visited him and his staff in 1918, during the World War, at Queen's Hospital in Sidcup, England, and again in London in 1927, as well as having read his book on plastic surgery, which is a classic, I feel sure that whatever he has to say about the subject can be relied upon. Therefore, when he made the pertinent statement that the majority of surgeons have the wrong slant on the indication for cosmetic surgery, I was compelled to take notice. I do not believe it is necessary for me to go into any detail to establish my interest and familiarity with the subject of plastic surgery, as I have done considerable work along this line, as far back as 1894, when the great Nicholas Senn was doing reconstructive plastic surgery and I had the pleasure of dressing some of his cases.

In 1908, I presented a series of cases of plastic surgery before the American Laryngological, Rhinological and Otological Society. Most of them were of the major reconstruction variety, although there were a few cosmetic cases among them. In my paper I asked for a frank discussion as to whether the members thought that this type of surgery was proper for our specialty, because I had heard that some men considered it questionable or bordering upon the unethical. One will note that at that time I expressed myself similarly to Dr. Gillies' recent utterances.

Permit me to quote from the transactions of 1908 of that Society: "This subject has been, and still is, to a great extent, treated by the general surgeon, notwithstanding the fact that detailed intranasal surgery is often necessary to obtain the best results. Again, a large majority of external nasal deformities are treated by charlatans be-

cause general surgeons, as well as rhinologists, often refuse and discourage treatment of the above named conditions for cosmetic purposes. This fact, I believe, is responsible for many bad results, accidents and malformations, rather than improvements. Many such patients belong in the care of a neurologist and should receive the strictest attention to prevent them from falling into the hands of an unskilled so-called specialist and beauty doctor.

"Those of you who have seen some of these cases after they were corrected by these so-called specialists wonder if you have not acted unwisely by refusing to listen to these unfortunate individuals."

Since 1908 I have refused to do cosmetic borderline operations in many instances, thus falling in that group of surgeons that Gillies criticizes. However, I have done a fair number of reductions and building up of parts about the head and neck that are decidedly cosmetic.

Today I am convinced that cosmetic surgery has come to stay and not only should we be willing to do the work but rather encourage it. There are so many unscrupulous men in this field that the earlier we let the public know we are doing this work the earlier will the poorly prepared advertising quack be driven out. When I say we should let the public know that we do this type of work I do not mean by press advertising or through any kind of lay literature.

One very important point is to make it possible for the average individual to have this work done. Thus far, like prosthetic dentistry, only the well-to-do can afford to have plastic operations performed. I believe that this is wrong and can be corrected. Many of these individuals will borrow to get money enough to have this work done, especially that most difficult individual, the psychoneurotic. Here is where one should make a sharp distinction as to whether to operate or not, because it has been my experience, as well as that of others, that no matter how good a result is obtained most of these individuals are never satisfied. However, I have seen cases where the work was so unsatisfactory that it is not at all surprising that the patients were dissatisfied. This occurs especially when the work is done by an occasional operator in plastic surgery.

While I hope that this work may become a regular rhinologic procedure and more widely spread, I believe plastic operations should be performed only by those who have prepared themselves in this field. Therefore, I would urge the establishment of clinics devoted especially to plastic surgery and only in our regular teaching institutions.

The technical side of plastic surgery is, of course, most important and new ideas are constantly presented. The literature, especially in the English language, is so voluminous that any reference on my part about the technic would surely be out of place.

I am confident that anyone interested in plastic surgery can obtain advice, instruction or even courses in plastic reconstruction or cosmetic surgery.

To be sure, such courses are not easily obtained at this time because most of the patients are seen in private practice and are somewhat supersensitive when demonstrated. Therefore, I repeat that there is a need of plastic surgery clinics in reconstructive as well as cosmetic operations within our teaching institutions.

Maliniak has just published a book entitled "Sculpture in the Living," which I have perused and found very interesting and instructive. The title appears quite "catchy," but since it is for the general practitioner and not the layman, I am sure it can do no harm.

What about the irregular plastic surgeon? Throughout this country there are men and women who place enticing advertising in the lay press magazines, in beauty shops and newspapers, and use many other less dignified yet nevertheless clever methods of attracting clients.

Two years ago a plastic surgeon told me that there were thirty-two individuals doing plastic operations in and about one city, and only two of these were members of the regular local medical societies. It is well for the officers of local medical societies to be on the lookout for some of these individuals who are attempting to obtain membership in the various societies for advertising purposes.

## DISCUSSION

WALTER H. THEOBALD, M.D., Chicago, Ill.: Doctor Beck has demonstrated the perplexing problems which confront the surgeon who contemplates reconstructive work on the head and neck.

The cases shown by him require patience and accuracy in operating, sometimes over and over again, in order to achieve results. The use of mechanical appliances is a tribute to his ingenious imagination. In those extensive defects where there is total or subtotal loss of parts and where plastic repair is altogether too laborious and time-consuming and perhaps objected to by the patient, the use of a prosthesis seems to me most satisfactory. This is particularly true when dealing with destructive lesions involving the palate, antrum or upper maxilla.

Another group of cases I wish to touch upon include injuries to the face and jaws from automobile accidents which have become an increasingly important problem. In emergency cases where fractures of the orbit, nasal bones or



upper maxilla may be present, it is important to make note of tooth occlusion. Any slight misplacement is usually manifested here. One important point in treating facial injuries is making a careful examination and diagnosis of the injury by direct examination and x-ray before any attempt at correction is made. Many bad deformities are the result of too hasty attempt on the part of one perhaps not qualified to correct these deformities or in too big a hurry to suture the skin laceration without finding out what goes on underneath. Unfortunately, this is often the case in giving first aid treatment. When such difficulties are to be dealt with, it is well to wait perhaps six months before doing a plastic repair.

Correction of bony misplacements may be attempted but not by open operation. These cases are usually seen several days to a week following first aid surgery. Lacerations have been hurriedly sutured with poor approximation and the results are not too comforting to the patient. Such facial plastic repair must be dealt with at a later date.

It is important to mobilize fractures of the nasal bones as early as possible. They are easily handled if treated in time and treated properly. Two weeks is about the limit in which one can expect to get a good result in a severe fracture. After that time there is callus formation and the bone fits more or less into the new position.

H. M. TRUSLER, M.D., Indianapolis, Ind.: Reconstructive and plastic surgery demand recognition and attention from the medical profession. They are and should be highly specialized branches of surgery, requiring a certain degree of natural aptitude for the work. But more especially, they require that the operator must have ample clinical material and special opportunities for the acquiring of special knowledge and technic. Many of the problems are extremely difficult. The operator who makes sporadic efforts to dabble in plastic surgery does an injustice not only to the patient, but to himself as well.

At the Indiana University Hospitals, Doctor Gatch has specified a separate division of plastic surgery. The following are typical examples of the problems with which we are confronted and solution of which we have accomplished by reconstructive surgery.

1. Burn scar deformities.

The scars which follow third degree burns may be very disfiguring or disabling depending upon the extent and location of the deformity. Many of these can be corrected only by pedicle grafts or other major reconstructive surgical procedures.

2. Neoplasms of the face.

X-ray and radium therapy have a place in the management of these conditions. Too often, however, these agents are improperly applied and the patient thereby loses the chance for the complete cure afforded by early surgical removal. In this field electrosurgery has much to offer.

Many times the lesion is allowed to advance to a point where surgical removal is mutilating, and for this reason any satisfactory management of such a case must rest upon plastic surgery. The determination as to whether any given lesion is surgically accessible, is a matter requiring surgical judgment based upon experience and special study.

3. Congenital defects.

In this group the problems most commonly encountered are cleft lip and cleft palate.

Good results require not only a close approach to normal appearance, but also provision for normal speech. In this phase of plastic surgery, Doctor Lingeman and I are collaborating.

#### 4. Traumatic defects and cosmetic problems.

Among the more important of these are the traumatic deformities of the nose and disfiguring scars of the face. In addition, however, there are congenital abnormalities of the nose and the ears, also other defects which are a source of concern to some individuals for purely cosmetic reasons. In the past, these problems have not been sufficiently recognized by ethical practitioners of medicine and surgery, so that as Doctor Beck has stated the unfortunate victim of a cosmetic defect has too often fallen into the hands of the charlatan who advertises himself as a plastic surgeon.

Patients who present these cosmetic defects either real or imaginary, can and should be properly advised. Many of them are of neurotic temperament or have been misinformed and deluded as to the possibilities of plastic surgery.

S. SALINGER, M.D., Chicago, Ill.: I would like to mention one or two things that have been of great help to me in doing minor plastic work around the lips and mouth. One is the planting of a Thiersch graft into the skin, sewing it and allowing it to remain, then opening it to get additional tissue to supply the area to be covered. The other is the use of cartilage taken from the back of the auricle to fill in minor defects in the bridge of the nose. It is simple, carries no risk, and does not involve the use of a foreign body or entail an extra operation for removing the costal cartilage.

JOSEPH C. BECK, M.D. (closing): It is time that more of the younger men got into this work. There are too many occasional operators—sincere and honest, but with insufficient training, and the reason is that there is not enough of this work in private practice. We need, as Doctor Trusler says, in every university a department of plastic surgery where poor people are taken care of. I have been unsuccessful in training men because they do not give it the time. They know the headaches I have had with some of these cases. But there is a certain number of men who will do the dignified work and excellent work that we must have.

# A STUDY OF BRONCHIECTASIS WITH REFERENCE TO ITS ETIOLOGY AND MANAGEMENT\*

By L. T. MEIKS, M.D.

Indianapolis, Ind.

These observations are based upon a study of seventy-one patients who for one reason or another were suspected of having bronchiectasis. In the majority of cases the chief complaint was a persistent productive cough, but in a few instances the presence of abnormal physical signs in the lung bases, or infiltration shown by a chest x-ray have led to an investigation demonstrating cavitation in patients who were not at that time coughing. All the patients were children, fifteen years of age or younger. Since bronchiectasis usually has its onset in childhood the study of young patients would seem to offer some advantage in any investigation of etiology, as less time has elapsed since the beginning of the process, and because there are, on the whole, fewer complicating factors to be considered. In five cases we had the opportunity of studying the patients in the hospital while the bronchiectasis developed as a complication of pneumonia, for which they were originally admitted. All patients had x-rays of the chest and sinuses, and practically all were examined by x-ray after the instillation of lipiodol into the bronchi. All were examined by an otolaryngologist.

Of the group of seventy-one patients, forty-nine were found to have definite cavities or dilatation of the bronchi. Of the remainder, eighteen were proven not to have any cavitation, although their symptoms and signs had strongly suggested that bronchiectasis was present. In four cases the study was not complete, and it was not definitely proven whether cavities were present or not. Since it is the opinion of some writers<sup>1, 2, 3</sup> that these chronic lung infections associated with productive cough, and diagnosed variously as chronic bronchitis, chronic pneumonia and fibrosis of the lung, represent essentially the same disease process as bronchiectasis, in an earlier stage, the eighteen cases without cavities have been included in the analysis. In a few instances the data in the charts were not complete, so that they were omitted in the tabulation of that particular item. The age of onset is shown in Table 1. It is apparent that the

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\* From the Department of Pediatrics, Indiana University School of Medicine, and James Whitcomb Riley Hospital for Children.

TABLE 1.—AGE OF ONSET

	<i>Bronchiectasis</i> Number of cases	<i>Chronic pulmonary in- fection without cavities</i> Number of cases
Since birth.....	1	1
1 or less.....	6	5
2.....	5	2
3.....	6	1
4.....	2	2
5.....	2	0
6.....	8	2
7.....	6	0
8.....	2	1
9.....	0	0
10.....	0	0
11.....	1	2
12.....	5	1
13.....	1	0
14.....	0	0
15.....	0	0
	45	17

onset of bronchiectasis is most frequent during the first three years of life, and also at the ages of six and seven. This corresponds to the frequency of acute respiratory infections during the very early years and again shortly after the child starts to school. In the cases without cavities this increase at the time of starting school is not present, although the number of cases is probably not enough to mean much.

A study of the duration of symptoms gave no particular information except that it showed that those cases without cavities may last a long time. In seventeen cases where the duration could be determined in cases proved to have no cavitation, nine had coughed for more than four years.

In Table 2 is given a tabulation of the diseases immediately preceding the development of the symptoms. In the cases where the difficulty was initiated by a specific infectious disease the usual story was that the child, having had the disease, failed to recover in the usual manner, but was left with a productive cough. Unless the acute disease merged imperceptibly into the chronic one it was not regarded as being the initiating factor. In the great majority of cases it was necessary to rely upon the history as given by the family as to the occurrence of the antecedent acute infection, since they were



TABLE 2.—DISEASES INITIATING SYMPTOMS

	<i>Bronchiectasis</i> <i>Number of cases</i>	<i>Chronic pulmonary in-</i> <i>fection without cavities</i> <i>Number of cases</i>
Pneumonia .....	11	8
Pneumonia and empyema.....	2	0
Pertussis .....	5	1
Pertussis and pneumonia.....	6	0
Pertussis, measles and pneumonia	2	1
Measles and pneumonia .....	4	0
Influenza .....	2	1
Influenza and measles .....	2	0
Influenza and pneumonia.....	1	1
Sinusitis .....	3	0
Asthma .....	1	0
None, gradual onset .....	10	3
Congenital .....	0	1
	—	—
	49	16
Bronchial or pulmonary disease	35—72.9%	12—75%

usually brought to the hospital long after the onset. However, the relationship was ordinarily so clear that there was seldom any doubt as to the accuracy of the original diagnosis. It is apparent from the table that the majority of cases, both of bronchiectasis and chronic pulmonary infection without cavitation, began as a complication of a disease where there is an inflammatory lesion of the bronchi, and in the cases of pneumonia, involvement of the alveoli in addition. If all the cases preceded by an acute infection are placed in one group it is found that 72.9 percent of the cases of bronchiectasis and 75 percent of the cases without cavities began with an acute disease. It is interesting to note that the percentage is practically the same in both groups of cases.

Evidence of coexisting nasal sinus infection was looked for in all cases. If the history indicated an unusual number of "colds," or if they had been unduly severe or protracted, it was taken to be presumptive evidence of sinus disease even though there was no evidence of any at the time of examination. Any evidence of abnormality on clinical examination and even slight haziness in the sinus x-rays were taken, at least for this tabulation, as an indication of sinus disease. Using these criteria it was found that 81.2 percent of forty-eight cases of bronchiectasis, and 66.6 percent of eighteen cases of lung infection without cavities had evidence of chronic sinus infection. In the majority of cases the evidence of sinus disease was

perfectly definite. The severity of the sinus disease did not bear any direct relationship to the extent and severity of the pulmonary lesion. In a few cases there was no indication of sinus infection when first seen, but on a subsequent admission, months or years later, out-spoken evidence of sinus disease was found.

In seven cases of bronchiectasis there was a clear history of the simultaneous onset of unquestionable symptoms of sinus infection and the pulmonary difficulty. Three of these are tabulated above as cases following sinusitis. The other four followed pneumonia, the parents having noticed that as the child began to improve from the more acute symptoms his nose became obstructed and a marked nasal discharge developed, and that these symptoms, in addition to the productive cough, had continued. The simultaneous appearance of sinus symptoms and productive cough also occurred twice in patients without cavities, once following whooping cough and once after pneumonia.

The location of the cavitation was determined entirely on the basis of visualization with lipiodol, since it was our experience, as well as that of others,<sup>2, 4</sup> that opinions based on physical signs or ordinary chest x-rays were apt to be quite unreliable. Not infrequently signs would be present on both sides, but actual cavitation only in one. This would seem to indicate that the two groups of cases being considered, that is, those with cavities and those without, are really very closely related, since the only means of separating them is by the use of lipiodol. It seems quite likely that if these patients were examined later that some of them would by that time have cavities on the opposite side, although so far, on the few patients completely reexamined, we have not had that experience.

In nineteen of the forty-nine cases of proven bronchiectasis the cavitation or bronchial dilatation was present on both sides to practically the same extent. In seventeen cases it was confined to the left, and in eight to the right side. In three it was marked on the left and slight on the right, and in two marked on the right and slight on the left. So, in thirty (61.2 percent) cases the dilatation was either predominantly or exclusively on one side, and of these thirty unilateral cases twenty (66.6 percent) were on the left. In all cases the dilatation was either confined to the lower lobes, or more extensive there than in the uppers. It was only in the most extensive cases that the upper lobes were involved at all, and no cases were seen where the process was confined to the upper lobes.

From this series of cases, as well as others<sup>1, 2, 3, 5, 6</sup> which have appeared in the literature, a few facts are apparent.

1. The onset is usually early in life, most often during the first three or four years, with perhaps an increase also in the frequency of onset at the ages of six and seven.

2. The lesion is predominantly in the lower lobes of the lungs.

3. It is more often confined to one side than not, and when it is, it is more often on the left.

4. The characteristic symptoms, that is, chronic productive cough with purulent sputum, the tendency to exacerbations following acute upper respiratory infections, the hemoptysis, the rather mild constitutional symptoms except during the occasional acute flare ups usually diagnosed as pneumonia, frequently follow acute infectious diseases, particularly influenza, pertussis, measles, and pneumonia, in which there is an invasion of the bronchial mucous membrane by the infecting agent.

5. There is frequently an associated infection, usually of a chronic nature, of the nasal accessory sinuses.

Any inquiry into the pathogenesis of bronchiectasis must take these facts into consideration. Reports of other series of cases indicate onset early in life, and especially during the first years. This would seem to indicate primarily the importance of pulmonary infections as an etiological factor. Pneumonia, particularly bronchopneumonia is preponderantly a disease of the first three years of life. Muir<sup>7</sup> believes that the cavities are produced by mechanical factors acting on bronchi which are weakened by disease, particularly when the bronchi are not free to expand. In regard to this hypothesis it is of interest to recall that small children with pneumonia frequently have considerable mechanical difficulty in breathing due to the partial obstruction of the bronchi and bronchioles by exudate and swollen mucous membrane and made evident by the marked retraction of the costal margin and sternum with each inspiration. Under such conditions the air pressure within the bronchi must be considerably increased, and might very well be instrumental in causing the formation of cavities.

The reason for the preponderant involvement of the lower lobes is not clear. The most obvious explanation seems to be that gravity is in some way concerned, and would fit in well with the idea that the aspiration of infected material from the nasal passages is an important etiological factor. On the other hand, if aspiration is the prime factor one would expect the lesion to be bilateral, or, if unilateral, to be preponderantly on the right. This is contrary to the facts since more than half the cases are involved on only one side, and in the unilateral cases more frequently on the left. Perhaps an

explanation which does fit the facts about localization is that if the process has been started by an infection of the bronchi or parenchyma of the lung the accumulation of infected secretions may prolong and make worse the process which has already been started. This would readily explain the preponderant involvement of the left lower lobe, from which material is expectorated with greater difficulty since it must take a somewhat more circuitous route before reaching the trachea.

That sinus disease is frequently associated is certain. Clerf<sup>8</sup> believes that sinus infections are present in a much higher percentage of the cases with bilateral involvement than in those with cavities on only one side. Our cases have not shown this to such a degree. While sinus infections were somewhat more common when both sides were involved, the difference was not great, and several cases with bilateral lesions had no evidence of sinus disease.

Davidson and Pearson<sup>2</sup> emphasize the conception of what may be termed a functional unity of the upper and lower air passages and believe that any infection involving either the upper or lower portion is apt to involve the other also, so that an infection involving the whole respiratory tract may initiate chronic changes in both the sinuses and lungs, and that each of these lesions may contribute to the persistence and severity of the other. In this connection it is interesting that in nine of our patients there was a definite history of simultaneous onset of sinus and chest symptoms, in most cases following pneumonia.

That chronic sinus disease has a deleterious effect upon the general health, and predisposes to repeated lower respiratory tract disease is too well known to merit comment. Likewise, there can be little doubt that sinus infections contribute to the severity and persistence of the involvement of the lungs.

Many articles<sup>1, 2, 3, 4, 5, 6</sup> on this subject have stressed the frequency with which bronchiectasis follows acute respiratory disease, especially pertussis, influenza, measles, and pneumonia. In the majority of cases it is certainly a complication of inflammation involving primarily the tissues of the bronchi and lung parenchyma. However, there are a considerable number of cases where there is no history of such antecedent disease. Davidson and Pearson<sup>2</sup> have suggested that possibly in these cases there has been a true but slight pneumonic condition in connection with a respiratory infection with very mild clinical manifestations, but of sufficient extent to lead to necrotizing lesions in the lungs. Certainly the extent of the bronchiectasis bears no direct relationship to the severity of the pneumonia



when such has preceded it. Another fact which perhaps lends some support to this view is that the severity of lower respiratory infections in children varies imperceptibly from the mildest sort of tracheitis to the most severe pneumonia, so that in many cases it is quite impossible to differentiate between severe bronchitis and mild pneumonia. The proof of this is of course lacking since such patients do not die.

The actual development of cavities is well shown in the study by McNeil, Macgregor and Alexander<sup>9</sup> of seven autopsies on patients who had recently had pneumonia. They were able to demonstrate all stages in the development of cavities, beginning with invasion and ulceration of the bronchial mucous membrane and leading to excavation of the surrounding lung tissue through the sloughing of infected tissue. This cavity later is partially filled in by granulation tissue and is eventually lined by fibrous tissue.

A correlation of the information we have concerning bronchiectasis makes it seem probable that the process in practically all cases is initiated by inflammation of the bronchi of such severity that there is an interruption of its continuity by ulceration, and that there is always an associated infection of the surrounding lung tissue. After the process is initiated the accumulation of secretions, especially when there is a continued activity of the infectious process, may add greatly to the damage. Mechanical factors, particularly cough and partial obstruction of the bronchi, is probably a contributing factor. The frequently associated sinus disease probably does not play an important part except in so far as it may predispose to inflammatory lesions of the bronchi. However, once the process is started, the presence of chronic sinusitis probably tends to make it worse. Perhaps the frequency of associated sinusitis is an expression of the vulnerability of the respiratory tract in general.

Treatment of these patients has been undertaken with three main objects in view: First, clearing up, in so far as possible, foci of infection in the upper respiratory passages, when any were present; second, the promotion of adequate drainage of the cavities; and third, rest.

Treatment of the infections of the upper respiratory tract has been carried out along the usual lines, always under the supervision of the consulting otolaryngologist. Tonsils and adenoids, if present, have been removed. In a few cases tonsillar tags remaining after previous operation have been excised. In several cases presenting no signs of upper respiratory tract disease the tonsils and adenoids have been removed, more or less on suspicion. The management of

the sinus infection depended, of course, on the findings in the individual patient. Single or repeated maxillary lavage was the most frequently form of therapy. In many cases maxillary windows were made. The results from these relatively simple therapeutic measures were not satisfactory in some cases, doubtless due in some measure to the fact that adequate follow up examinations and treatment were impossible, since many of the patients lived so far from the hospital that frequent return visits could not be made. In recent years we have used the more radical Caldwell-Luc operation in those cases which had definite thickening of the mucous membrane as demonstrated by lipiodol injections into the maxillary antra. This operation has been done in ten cases, with definitely unsatisfactory results, both in regard to the sinus disease itself, and the bronchiectasis. Seven of these cases have been followed and in no case has there been more than a very moderate improvement. In some cases the nasal mucous membrane has been repeatedly shrunk by means of nose drops or topical applications with moderate benefit in some cases. Ethmoid exenteration, removal of nasal polyps and submucous resection were each done in one instance to meet particular indications in those patients. In general it has seemed that the treatment of the cases having outspoken sinus disease has been disappointing. Perhaps there is something about the respiratory mucous membrane, both in the nose and lungs, which renders it unduly susceptible to chronic inflammation, and unusually resistant to therapy. Certainly these patients with sinus disease have not responded to treatment of the sinuses as well as other patients with sinusitis not associated with bronchiectasis.

The adequate emptying of the cavities has been attempted chiefly by postural drainage. This was usually carried out in the ordinary fashion by having the patient hang over the side of the bed, with the hands resting on the floor. The exact position assumed depends upon the location of the cavities, and in some cases it has seemed helpful to have them lie with the affected side uppermost before assuming the head-down position. The frequency and duration with which this drainage has been carried out has varied with different patients, but has been usually from five to fifteen minutes three or four times a day. It should be frequent and long enough to prevent great accumulation of pus in the cavities. If the assumption of the drainage position results in the expectoration of very much sputum it is evident that the drainage is not being done frequently enough. In a few cases continuous postural drainage by the method suggested by Boyd<sup>6</sup> has been used. This device is essentially a Bradford frame upon which the child lies with the foot of the frame elevated to an

angle of 30 or 40 degrees. With this it is possible to keep the cavities practically empty at all times, and in some cases its use has resulted in marked improvement. It is really surprising how little discomfort these children have while lying continuously in a head-down position.

Many of the patients, especially during the past few years, have been bronchoscoped at least once. This has been done partly for diagnosis, partly in the search for foreign bodies which may have started the process and partly for treatment. In several cases growths of granulation tissue have been found in the bronchi or around the openings of the saccular cavities which have partially obstructed the drainage of these cavities. The removal of this granulation tissue has always resulted in much better drainage. In addition, those patients who with postural drainage are unable to dislodge the thick tenacious sputum which fills the cavities are usually greatly benefited by the aspiration of the sputum through the bronchoscope. Many of our patients have been repeatedly aspirated in this way, usually with very distinct benefit. Not infrequently it has been found that after a few aspirations the sputum becomes thinner and less tenacious, so that it can then be coughed up without the aid of the bronchoscope. Several times patients, after a bronchoscopic treatment, have asked to have it repeated because they felt so much better after it. When a child requests such an admittedly uncomfortable procedure, there certainly must have been considerable subjective improvement.

In seven cases where the cavitation was extensive on one side with a normal or only slightly involved lung on the other side, phrenic neurectomy has been done in an attempt to partially collapse the affected lobe. Since we believe that this is an operation not to be undertaken lightly, it has been done only on patients who were greatly incapacitated by the disease. Since these patients were also treated by postural drainage, and usually by repeated bronchoscopic aspiration, it is somewhat difficult to estimate the value of this procedure. However, the six of them who have been followed have done remarkably well, and it would certainly seem that, at least for this particular type of patient, the operation results in very decided improvement.

Neither pneumothorax, thoracoplasty, nor lobectomy has been used in any case. In a few cases autogenous vaccine has been used with no apparent benefit. Two patients felt distinctly improved following the diagnostic lipiodol injection, and it has been repeated many times in each of them at their request, perhaps with some benefit.

In this series it has seemed evident that the most important factor in the treatment is the maintenance of adequate drainage of the infected cavities. In general those who have followed instructions have tended to do much better than those who did not. Bronchoscopy has been of great aid in promoting drainage in cases where the sputum is tenacious or is partially obstructed by granulation tissue. In certain cases phrenic neurectomy is apparently of value. When present, the sinus disease should of course be treated, since improvement is frequently followed by improvement in the lung infection.

Of the forty-nine cases of bronchiectasis thirty-eight have been seen again, most of them a year or more after the first admission. Of these five were not improved in any way. Fourteen were moderately improved, by which it is meant that the cough was considerably less but still present to a considerable degree. Fifteen were found to be greatly improved. All these were practically free of productive cough. Some of them would have a productive cough for a week or two following an acute respiratory infection, but very little at any other time. A few of them were completely free of symptoms, but were not regarded as well because they were not studied with lipiodol to prove the disappearance of the cavities. Four patients are completely well, with no symptoms or abnormal physical signs, and with a complete disappearance as shown by lipiodol of the cavities which had previously been present. In two of these cases the cavities had previously been large but present only on one side. Of course, the follow up time on many of these who were improved is not sufficient to be at all sure of the ultimate outcome, and it seems likely that the difficulty will recur in many of those who were temporarily so definitely better. Nevertheless, definite improvement has been so frequent that we can now approach these patients in a fairly hopeful frame of mind.

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## DISCUSSION

HOWARD C. BALLENGER, M.D., Chicago, Ill.: Doctor Meiks' painstaking study and conservative conclusions demand careful and thoughtful consideration as it is such reports as this that settle ultimately the question concerning the rôle the nasal sinuses play in bronchiectasis. This association of sinusitis with these cases of chronic lung infection in such a high percentage of cases does not seem to be just an accident but is a matter of importance either as a secondary manifestation of the pulmonary disease or possibly as an etiological factor that should not be overlooked.

Perusal of the recent literature discloses the fact that the vast majority of opinion accepts this relationship and considers the care and treatment of bronchiectasis incomplete without careful investigation and treatment of the involved sinuses.

The incidence of chronic sinus disease in bronchiectasis varies with different investigators from 55 to 100 percent. Clerf in a recent report found an incidence of 82 percent, Kern and Schenck, 80 percent with the controls in their series showing an incidence of 26 percent. Doctor Meiks in this investigation reports an incidence of 81 percent at some time during the course of the bronchiectasis.

As brought out by Doctor Meiks in his paper, the factors that would indicate the sinuses are not the chief etiologic agent are:

1. The early age of onset of the bronchiectasis, that is during infancy and early childhood when chronic sinusitis is not so prevalent as later in life.
2. The frequency with which bronchiectasis is preceded by an acute infection involving the bronchi or alveoli.
3. The tendency of the lesions to be unilateral and preponderately on the right side.

The factors which would favor the theory that the sinuses are an important etiologic agent are:

1. The high percentage of patients with bronchiectasis in which sinusitis is present.
2. The known tendency of sinusitis to produce and prolong infections of the lower respiratory tract.
3. The preponderate involvement of the lower lobes in bronchiectasis which would be expected if due to aspiration or gravitation of infected material from the nasal passages.
4. The possibility that long continued flooding of the lymph channels from the sinuses produces a bronchiectasis. Mullin injected ink into the maxillary sinuses of rabbits and recovered the ink from the bronchial glands.

It is possible, as found in seven of Doctor Meiks' cases, that there is a simultaneous onset of sinus and chest symptoms.

Inasmuch as the sinus may be an etiological factor of importance and in any event a contributing agent that very likely would prolong, or enhance the chest

pathology, it is essential that the rhinologist give attention to the sinuses in the hope of giving some relief to the bronchiectasis.

WILLIAM V. MULLIN, M.D., Cleveland, Ohio: I wrote my thesis in this Society in 1919 on this very subject, and then a few years ago before the American Laryngological Association I gave a follow-up of a group of cases that I had followed for thirteen years. In other words, I followed them from infancy to young adult life. I am perfectly willing to listen to all this, but I do not think there has much been added to what I had at that time. It is not just right of Doctor Meiks to speak offhand of "sinus disease." I think we have to classify sinus disease. I have said over and over again that there are some types of sinus disease that do not produce trouble in the lower respiratory tract, and others that do. Doctor Galloway spoke of foreign bodies. I showed a case where an aneurysm pushed into the bronchus and blocked it and produced bronchiectasis. Anything that produces obstruction of the bronchus and blocks the peristaltic flow will produce dilatation below.

The doctor spoke of cleaning the polyps out of the ethmoid and eliminating all the secretion running down from the nose into the lower respiratory tract. There is where classification must come in. Polyps in the ethmoids may be nothing more than allergy. A mucous polyp in a majority of instances is probably an allergic condition and has nothing to do with the type of case you are talking about today. But if you have a chronic purulent infection in the sinus, very likely you will have an infection in the lower respiratory tract. If you wish to refer to the pathology that occurs down there, I give it in a number of experiments I did with India ink dropped into the nose of rabbits and which ran down into the lower tract.

As to treatment on a Bradford frame, right after I came out of service I took an ordinary carpenter's horse and made a bar across it which was longer at one end than the other, and put those children on it, lying down. That is how long I have been using postural drainage for these children, and I am glad to say a good many of them are now in young adult life and are well.

As to the relationship between acute infectious disease and bronchiectasis I mentioned that, too. You must remember that bronchiectasis is not produced overnight, in a week, or in a month. It is bound to come over a long period of time, and if you have bronchiectasis associated with measles or some of the acute infectious diseases you get a sinus disease, and then about a year afterwards, and not much less than that, you will have a bronchiectasis, but by that time the sinus disease may be well. It may have been the start, but these other processes carry it on.

SAMUEL IGLAUER, M.D., Cincinnati, Ohio: I am very much interested in this subject, not so much from the standpoint of a pediatrician as in the various methods of introducing iodized oil into the tracheobronchial tree. I talked on this in Indianapolis some six years ago.

One point I wish to mention is the presence of cavitation on the left side. There is an area behind the heart of lung tissue which is frequently involved and shows much cavitation only after the injection of lipiodol. In this area the heart sounds overlap the pulmonary sounds. Probably the lung is mechanically interfered with in the expulsion of purulent material.

Another thing that has interested me in doing bronchoscopies, especially in adults, is that if you examine a patient who has not been posturized that

morning you will find the lungs full of pus; if the patient has been posturized you will find the tracheobronchial tree entirely clean, so that by mere visual control you can see how readily posture evacuates these cavities. I remember one boy for whom I prescribed a trapeze, and his father would take him out each morning and let him hang on it and in that way get rid of the bronchial secretions by centrifugal force, and he had a good time doing it.

I had an interesting experience the other day which shows the possibility of bronchial obstruction. I had an infant seventeen months old on whom I expected to do a mastoid in the afternoon. In the morning I took a picture of the chest and found a transposition of the mediastinum to the left. That informed me that this child evidently had a bronchial block. They took the child down stairs and fluoroscoped him and the lung was back in position. I believe that indicates that occasionally we have a blocked bronchus and do not know anything about it. However, I was not going to give this child inhalation anesthesia, so I did the operation under local.

D. O. KEARBY, M.D., Indianapolis, Ind.: Many of these cases reported by Doctor Meiks have been properly bronchoscoped. We are rather systematic at the University Hospital in regard to cases that come in, as to history, general physical examination, and then roentgen examination. The majority of these cases come to the bronchoscopic department last. Doctor Jackson talks about foreign bodies being a cause, and there is no question about that, but we have only found one case that had a foreign body as the cause. We have found in two cases some strictures rather well developed due to bronchial compression by a large gland on the outside. In one case we found a benign tumor obstructing, but most common are these masses of granulations which we find with the bronchoscope as a part of the treatment. It is spectacular to see these cases clear up after aspiration and topical treatment. We had one case in which we recovered tuberculosis organisms that had not been diagnosed, and another in which we found Vincent's organisms.

In our study of these cases we are fairly well convinced that the sinus is not the primary cause. Every laryngologist knows that every child with maxillary sinusitis has a cough.

LYMAN MEIKS, M.D. (closing): I think the question of the relation of the sinuses to chest infection certainly is not settled.

As far as foreign bodies are concerned, we have suspected their presence many times, but so far have not discovered any. Certainly if discovered they would be a very patent source of bronchiectasis. As to obstructions such as aneurysms, obviously if the lung is atelectatic that is a source of development of cavitation.

In regard to aspirating pus from the nose into the lung, this occasionally does happen, but if the bronchi are normal they are able to eliminate the material without the development of disease. It seems at least that before that can enter into the picture there must have been some preceding damage to the bronchi or the lung itself.

In regard to the tubes of Doctor Iglauer, he took some little trouble to get them for us and we have been using them with much success. We have better films in that way than by any other method.

## SURGICAL APPROACHES TO DEEP SUPPURATION IN THE NECK AND POSTERIOR MEDIASTINUM\*

By SAMUEL IGLAUER, M.D.

Cincinnati, Ohio

In his classic treatise entitled "Topographical Anatomy," published in 1887, Tillaux stated that the cervical fascia is an exceedingly variable structure and that this variability explains a certain confusion or difference in the descriptions of various authors. It was Tillaux's chief endeavor to present a description of the cervical fasciæ which would harmonize with the recognized pathologic conditions of the neck. About forty years later Tandler,<sup>1</sup> after mentioning the complicated arrangement and interrelations of the various structures of the neck, pointed out that this very complexity led to the most divergent descriptions and interpretations by various anatomists.

The cervical fascia is usually described as consisting of three layers—a superficial or investing, a middle or visceral and a deep or prevertebral layer. The first or outer layer completely surrounds the neck and is attached to the skull and mandible above and to the clavicles and sternum below. The second or visceral layer invests the larynx and trachea in front and extends laterally and posteriorly to include the pharynx and esophagus. It splits to embrace the thyroid gland. The visceral layer is attached to the base of the skull above and behind and is continuous with the mediastinum below. If the cervical viscera are removed from a cadaver a space remains which is known as the visceral compartment. The third or deepest layer, known as the prevertebral fascia, extends across the neck immediately in front of the vertebral column and unites laterally with the two other layers. At the point at which the three layers come together they form a so-called sheath for the great vessels of the neck, the perivascular sheath.

Certain spaces exist between the fascial lamellæ. The important visceral compartment has already been mentioned. The loose areolar tissue between the pharynx and esophagus in front and the prevertebral fascia behind constitutes the postvisceral space. The previsceral or interaponeurotic space is situated between the trachea behind and the sternohyoid and sternothyroid muscles in front.

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Recent studies by Weintraub<sup>2</sup> and Batson<sup>3</sup> present a new interpretation of the anatomic relations and structure of the cervical connective tissues. These authors considered the neck as made up of two parts—the cervix proper, *i.e.*, the cervical vertebræ together with the postvertebral muscles, and, second, the gula or visceral parts of the neck with their associated muscles, blood vessels and nerves. The visceral part of the neck is of especial interest to the surgeon. Doctor Weintraub<sup>4</sup> demonstrated the technic of the anatomic dissection on which the study of this portion is based. The head and neck are split in the middle line, and transverse sections of the neck are also made. The tissues are not dissected but are merely teased apart beginning at the spinal column. This method demonstrates that the connective tissues of the neck are arranged in two groups, one of which is termed pedicles and the other telæ, or weblike tissue spaces.

The pedicles consist of sheets or lamellæ containing the supply of blood vessels and nerves to the structures in which they terminate. The pedicles may be likened to the mesenteries of the abdominal viscera. The chief cervical pedicles originate from the neurovascular bundle, from which in transverse section they are seen to radiate in a manner similar to that of the ribs of a folding fan. The neurovascular bundle, or hinge of the fan, has the form not of a conduit but of a nutrient stalk. The telæ, represented by the webbing of the fan, consist of loose areolar tissue lying between the various pedicles and are coextensive with the so-called tissue spaces of the older anatomists. There are no macroscopic blood vessels within the telæ. The telæ are best developed in the areas in which the adjacent structures, for physiologic reasons, require the greatest freedom of movement. The technic of blunt separation discloses numerous spaces not demonstrable in ordinary dissections. For examples, the so-called retropharyngeal space consists not of one but of three distinct compartments—one large median compartment and a juxtamedian space on each side. A potential space also exists between the anterior spinal muscles and the prevertebral fascia on each side and continues downward behind the dome of the pleura (Figs. 1 and 2). In addition, the mucous membrane on the posterior wall of the pharynx can be elevated, disclosing two submucous spaces separated from each other by the median raphe (Fig. 1, 4).

Another space is located between the trachea and the esophagus, and two telæ lie between the depressor muscles of the larynx. A knowledge of these additional spaces or telæ is of considerable value in the study of infectious processes in the neck.

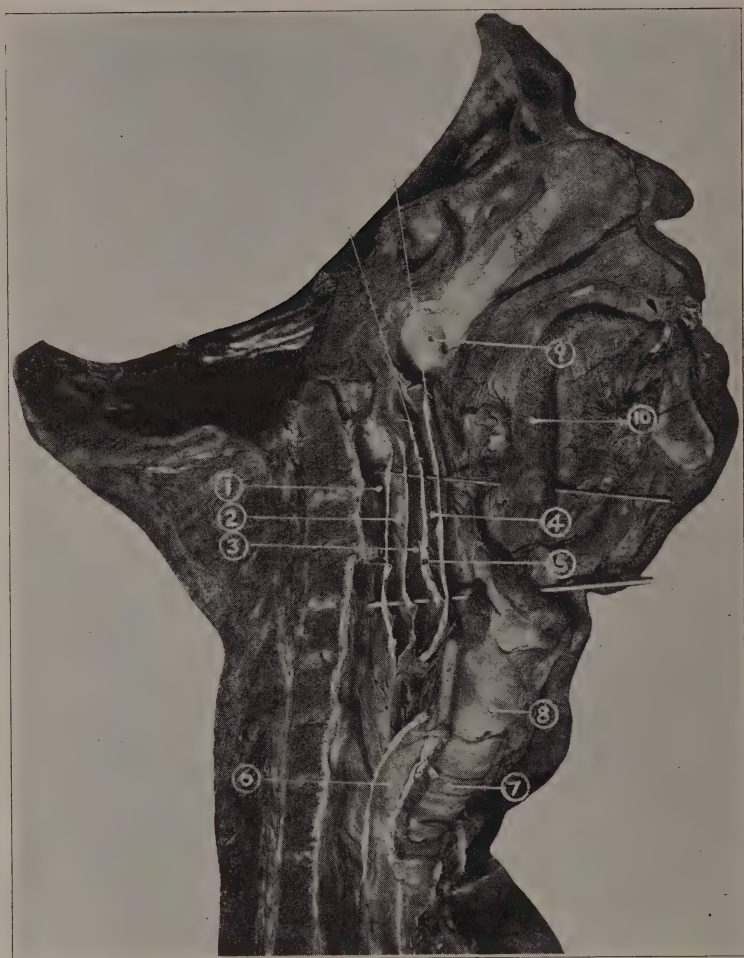


FIG. 1. Median section showing the left half of the head and neck. 1 indicates the left prevertebral space; 2, the median postvisceral space; 3, the left posterior peripharyngeal space; 4, the pharyngeal submucous space; 5, the muscle of the posterior wall of the pharynx; 6, the esophagotracheal space; 7, the trachea; 8, the larynx; 9, the torus tubarius with the ostium; and 10, the tongue.

### DEEP INFECTIONS

Abscesses or suppurations develop within the telæ and are limited by the pedicles, which one must either separate or perforate in approaching and draining the infected areas. Suppurative disease may, of course, break through the pedicles and extend from one tela to another. In this connection, I have demonstrated on the fresh cadaver the retropharyngoesophageal pathway of infection by packing the postvisceral compartment with lead gauze introduced into

it either from above or from below. The gauze is demonstrable in the roentgenogram, and it also delineates the space in vertical or horizontal sections of the frozen specimen. In Figure 3 the gauze

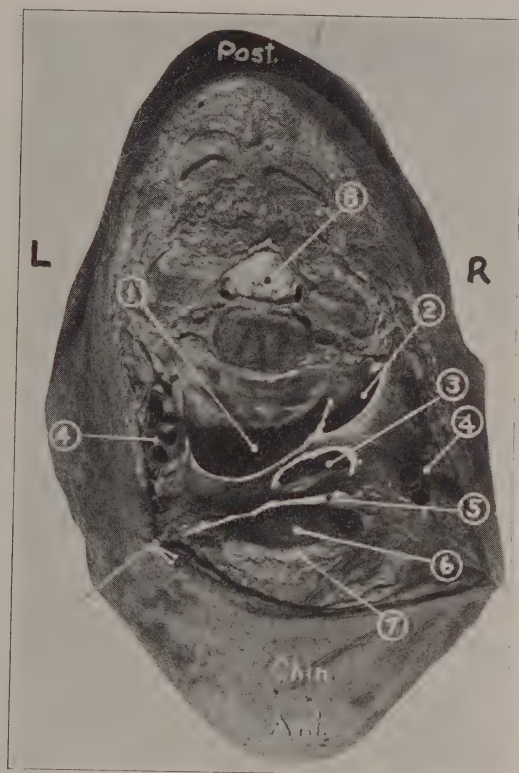


FIG. 2. Horizontal section through the neck at the level of the body of the fourth cervical vertebra, seen from below. 1 indicates the median postvisceral space; 2, the right prevertebral space; 3, the right posterior peripharyngeal space; 4, the neurovascular bundle; 5, the muscle of the posterior wall of the pharynx; 6, the cavum pharyngis; 7, the epiglottis; and 8, the spinal cord. Spaces on the left side corresponding to spaces 2 and 3 have not been opened.

pack is shown in place, and the vertical pathway from the base of the skull into the posterior part of the mediastinum is demonstrated. It is evident in Figure 4 that the lateral limits of the pathway are formed by the neurovascular bundle.

In addition to the present improved understanding of the anatomic relations of the neck and the usual methods of physical examination, there are two aids in the diagnosis of the presence, and in the determination of the site and extent, of infections of the neck. I refer to the use of the roentgenogram and of the esophagoscope. By



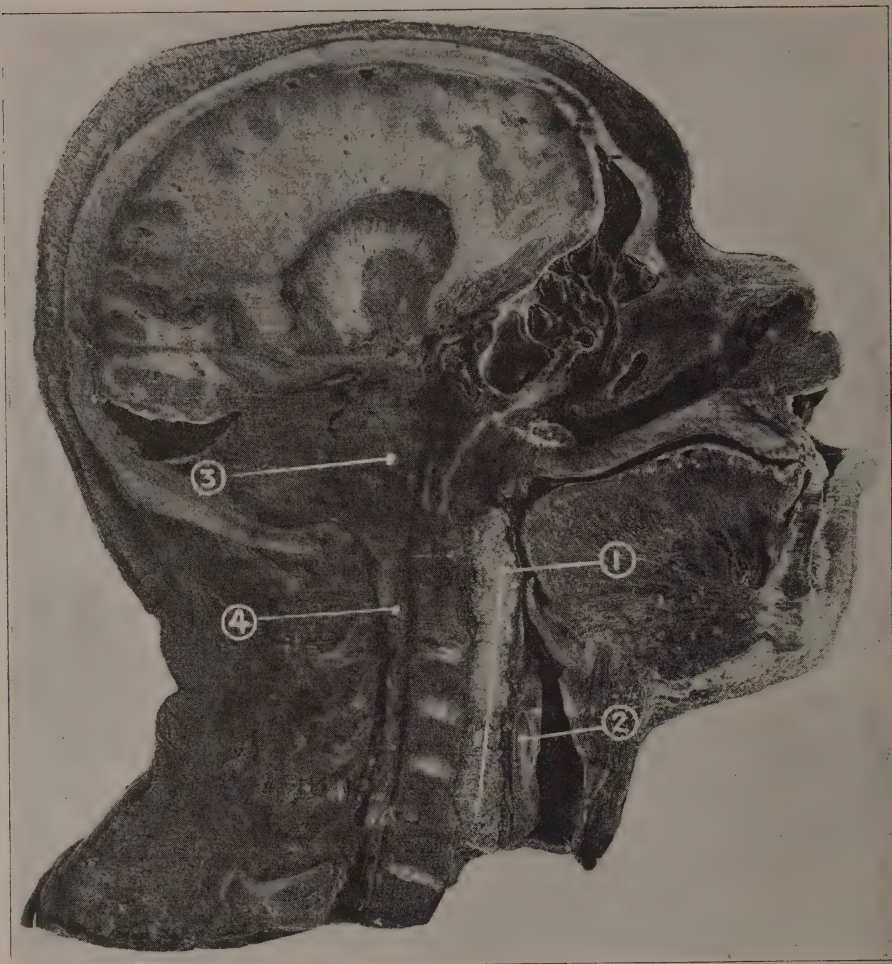


FIG. 3. Median frozen section showing the left half of the head and neck of an adult. 1 indicates the gauze pack in the postvisceral space; 2, the cricoid cartilage; 3, the brain stem; and 4, the spinal cord. The space filled by the gauze pack corresponds to space 2 in figure 1 and to space 1 in figure 2.

means of roentgenograms taken in the lateral and anteroposterior positions the swelling can usually be delineated both in the neck and in the mediastinum. In perforation of the esophagus interstitial emphysema about the site of perforation can also be demonstrated on the film, even when emphysematous crackling is not palpable at the root of the neck (Iglauer and Ransohoff<sup>5</sup>). If the patient is given iodized poppy-seed oil to swallow the opaque oil may pass through the perforation and appear outside the esophagus.



The introduction of an esophagoscope will reveal the site of greatest swelling as well as the presence or absence of a perforation.

#### RETROPHARYNGEAL INFECTION

Retropharyngeal abscess, resulting as a rule from the breaking down of an infected lymph gland, should usually be opened through

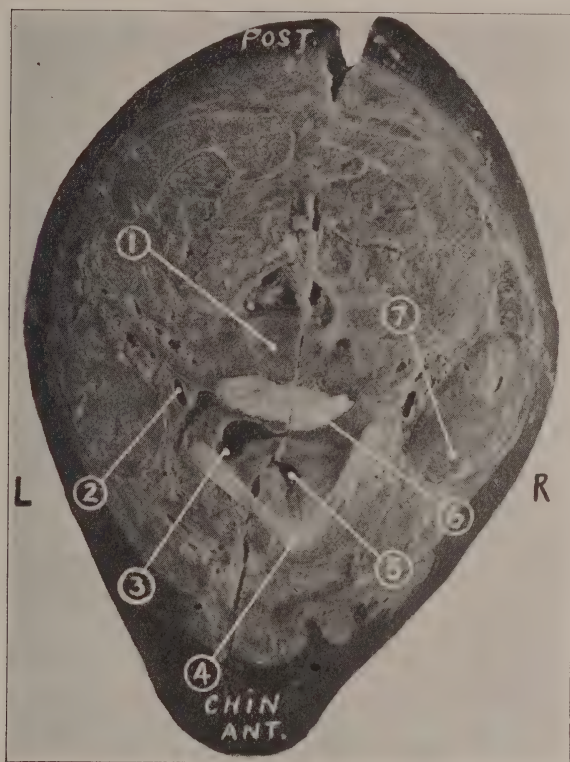


FIG. 4. Frozen cross-section of the neck of an adult at the level of the thyroid cartilage, view from below. 1 indicates a vertebral body; 2, the carotid artery; 3, the esophagus; 4, the thyroid cartilage; 5, the vocal cord; 6, the gauze pack in the postvisceral space; and 7, the sternocleidomastoid muscle.

the mouth. The patient should be placed in the hanging head position to prevent the aspiration of pus when the abscess is evacuated. With the index finger as a guide the bulging posterior pharyngeal wall should be perforated with a forceps, which should be opened within the cavity of the abscess. If a suction pump is available, it should be used to evacuate the pus from the pharynx.<sup>6</sup> Tucker<sup>7</sup> recommended opening the abscess through a pharyngoscope and aspirating the pus with a suction pipe introduced through the phar-

ngoscope. In cases of dyspnea he inserted a bronchoscope before using the pharyngoscope.

An external approach is indicated when the infection is secondary to spinal caries, in order to prevent contamination of the abscess with the buccal flora. Very large abscesses and retropharyngeal cellulitis should also be approached from the outside.

*Posterior Approach.*—By making the incision behind the sternocleidomastoid muscle practically all the branches from the carotid artery and the internal jugular vein can be avoided, and this usually renders the posterior approach much simpler than the anterior. Through a cutaneous incision the investing fascia is divided behind the muscle at a point on a level with the abscess. The patient's head should be rotated to the opposite side in order to draw the neurovascular bundle away from the spinal column. With blunt-pointed scissors the retropharynx can be reached and drained after perforation of a second layer of fascia situated behind the carotid sheath. In this approach the cervical ganglions, which are attached to the prevertebral fascia, should be avoided.

*Anterior Approach.*—This operation was originally described by Marschik,<sup>8</sup> of Vienna, under the name "superior cervical mediastinotomy" or "collar mediastinotomy." It was designed to drain infected areolar spaces about the lower part of the pharynx and the upper portion of the esophagus and to block the descent of infection through the upper thoracic aperture into the mediastinum. The operation was described by Schlemmer,<sup>9</sup> Glogau,<sup>10</sup> Palmer,<sup>11</sup> Beck<sup>12</sup> and others, and was recently modified and elaborated by Heatly and Pearse.<sup>13</sup> The incision as originally employed by Marschik extended along the entire anterior border of the sternocleidomastoid muscle, but Pearse found that the lower half of the incision usually gives a sufficient exposure.

The anterior border of the sternocleidomastoid muscle, together with the neurovascular bundle, is retracted laterally. The thyroid gland is exposed and retracted medially. The middle thyroid vein is ligated and divided, and the inferior thyroid artery is usually severed between ligatures. The omohyoid muscle is severed if necessary. By blunt dissection behind the thyroid gland the esophagus is exposed and lifted away from the prevertebral fascia. A gauze pack is inserted behind the esophagus and brought out through the lower angle of the wound. Heatly and Pearse stated: "If the symptoms point to unilateral involvement, the operation is terminated. If bilateral extravasation is suspected, a similar incision is made on the opposite side and a drain inserted down to meet that already

placed. Thus the visceral space is encircled and all paths of spread to the mediastinum effectively blocked."

Through a similar incision, preferably on the right side, Furstenberg<sup>14</sup> exposed the sternohyoid and the sternothyroid muscle and retracted these structures laterally, together with the sternocleidomastoid muscle and the underlying vascular bundle. The trachea and esophagus were then exposed, and by blunt dissection the posterior part of the mediastinum was entered and a drain inserted. Suction was applied at frequent intervals. In this approach the recurrent laryngeal nerve on the right and the thoracic duct on the left must be avoided.

In cases in which the infection has descended beyond the point at which the measures described are effective, dorsal mediastinotomy may offer the only hope of saving the patient. This formidable operation belongs in the domain of thoracic surgery. As a suggestion of a possible alternative to this procedure it may be stated that in several cases Seiffert<sup>15</sup> succeeded in draining a periesophageal infection into the esophagus by internal esophagotomy performed through an esophagoscope.

#### APPROACH TO THE PHARYNGOMAXILLARY FOSSA

Occasionally a case occurs as a sequel of tonsillectomy, under which circumstance the abscess may sometimes be reached by perforation of the constrictor muscle and its investing fascia through the bed of the tonsil.

Batson<sup>3</sup> demonstrated a simple external approach. A small vertical incision is made just above and behind the angle of the jaw; the fascia is divided, and the parotid gland is retracted backward. Scissors, curved on the flat surface, are introduced in a median forward direction, hugging the internal pterygoid muscle, opposite to which the abscess is to be entered.

In the cases in which the symptoms suggest thrombophlebitis a wide external approach, combined with an exposure of the internal jugular vein, is indicated. A curvilinear incision is made corresponding to the outline of the digastric fossa. The fascia is divided, and the submaxillary gland is exposed and partially elevated from its bed. The tip of the great horn of the hyoid bone is palpated; this, as described by Mosher,<sup>16</sup> constitutes an important landmark. The stylohyoid muscle and the posterior belly of the digastric muscle are exposed and retracted, and the parapharyngeal space<sup>16a</sup> is entered by blunt dissection. The posterior facial vein and its tributaries are

traced toward the tonsillar bed, and the jugular vein is laid bare to determine the downward extension of the thrombus. Diseased portions of the vein and its tributaries should be excised. The tonsil on the involved side should then be removed.

In a recent article Kramm<sup>17</sup> described what he considered a more accurate approach, affording better visualization of the cavity of the abscess and the tonsillar veins. The operation consists of an incision in the retromandibular fossa along the anterior border of the sternocleidomastoid muscle. The fascia is divided, exposing the lower posterior border of the parotid gland, which is avoided. The sternocleidomastoid muscle is retracted backward, exposing the posterior belly of the digastric muscle and the styloid process. The internal jugular vein lies parallel to the styloid process and behind it. The tendon of the styloglossus muscle is detached from the styloid process in front, and a long nasal speculum is inserted into the prestyloid compartment of the parapharynx. To reach the retrostyloid compartment the jugular vein is drawn forward and the posterior belly of the digastric muscle, backward. A speculum is then inserted into the posterior compartment.

#### APPROACH TO THE NEUROVASCULAR BUNDLE

Infection occurs either in the lymph glands adherent to the neurovascular bundle or in the form of a septic thrombus within the jugular vein or some of its tributaries. As already stated, the neurovascular bundle is not a tube or conduit, since it is not surrounded by any continuous lamellar fascia (Figs. 2 and 4). Parsons<sup>18</sup> regarded the "carotid sheath" as an artefact made with the scalpel by the anatomist or surgeon. The approach to the neurovascular bundle is usually made in the classic manner under the anterior border of the sternocleidomastoid muscle, and the bundle is exposed after retraction of the muscle. At times it may be advisable to split the muscle along the course of its fibers, and rarely it may be partially divided transversely. If the vein is seen to be occluded (or collapsed) it should be uncovered, together with its tributaries, until a normal area is reached. After ligation the diseased vein or veins should be resected.

#### LUDWIG'S ANGINA

No discussion of infections of the neck is complete without mention of Ludwig's angina. This clinical entity, an infection of the floor of the mouth, has been recognized for almost a century, since



Ludwig described the disease in 1836. So many excellent treatises have been written on the subject that there is but little to add. The rules for treatment are the same as those for most cervical infections—free incisions and drainage.

### SUMMARY

A brief review of some of the more recent studies of the cervical fasciæ is presented.

The surgical approaches to the various areolar spaces of the neck are described. The spaces under consideration include the retropharyngeal, the retroesophageal, the posterior mediastinal, the parapharyngeal, the neurovascular and the submental.

Prof. E. F. Malone and Dr. L. O. Morgan of the department of anatomy placed the material and facilities for this study at my disposal, and Dr. P. Goland assisted in some of the dissections.

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- 16a. Some confusion seems to arise from the terms "peripharyngeal" and "parapharyngeal," which are not interchangeable. The following quotation from one of my previous papers will aid in clarifying the subject:

"The parapharyngeal space, or the pharyngomaxillary fossa, is of great surgical importance. On cross section of the neck at the tonsil level, the fossa is triangular in outline. It is bounded mesially by the lateral pharynx wall, laterally by the horizontal ramus of the jaw, the internal pterygoid muscle, and the parotid gland, posteriorly by the prevertebral fascia. The styloid process and styloid muscles divide the space into an anterior and a posterior compartment (pre-styloid and retrostyloid). The posterior compartment contains the neuro-vascular bundle. The parapharyngeal space extends upward to the base of the skull and, according to Batson,<sup>9</sup> is limited below by the capsular fascias of the neck and the mylohyoid muscle.

Owing to its close proximity to the tonsil bed, infection of the parapharynx sometimes occurs as a complication of a peritonsillar or a retrotonsillar abscess."

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## DISCUSSION

S. SALINGER, M.D., Chicago, Ill.: I wish to mention two points. One is the existence of this posterior compartment in the pharyngeal space. In most of the old anatomies I could find no such compartment, although some authors mention it. It is evident, therefore, that it is not a constant structure, and I would be interested to know whether there are any other references outside of the work Doctor Iglauer mentioned.

Another thing is Kramm's approach to the pharyngeal space. The only objection I can see is the possibility of coming into contact with the facial nerve before it enters the parotid gland.

D. O. KEARBY, M.D., Indianapolis, Ind.: I would like to cite two similar cases in one of which a child swallowed a pork bone which penetrated the esophagus posteriorly. The bone was removed, but there was a cellulitis. It led to abscess formation posterior to the esophagus. The pus in that retro-esophageal space pushed up rather than down, lifting up the mucous membrane from the back wall of the pharynx. We treated that abscess with the laryngoscope and aspiration through a tube after which the whole process subsided.

The other case was that of an adult who swallowed a piece of rabbit bone. There had been two hours' work done on him trying to find the bone when he came to us. He had a cellulitis and some temperature, thought to be due to a retropharyngeal abscess. Incision failed to reveal any pus. This spicule

of bone was so small that the anteroposterior x-ray was obliterated by the spine and the bone could not be located. Three rather deep incisions were then made above the esophagus in the hypopharynx. An x-ray after the operation indicated that we had been near the bone because there was an air bubble. The swelling and temperature subsided, and in a week the man was feeling better, but the bone was still present. He refused further treatment. This was a year ago last October. I discussed that problem with all the surgeons and had as many different opinions as surgeons. I examined this man six weeks ago and found him well, but I presume he still carries the bone.

SAMUEL IGLAUER, M.D. (closing): Iodoform gauze is used as a drain. In regard to Kramm's approach, the styloid is the guide, just as in Mosher's approach it is the hyoid bone. Behind the styloid is the jugular vein, which you would have to separate. The Batson approach is very simple and probably would suffice in a case where you do not have sinus thrombosis. I use a speculum to look at the veins to see if they are thrombosed.

Replying to Doctor Galloway's question, emphysema after manipulation of a foreign body in the esophagus usually means perforation and if there is time enough it should be opened and drained. You probably could see the perforation if you go down again. If you find perforation then you know you have mediastinal infection or periesophageal infection.

In regard to Doctor Kearby's case, Rucker has described a few cases he has opened in which he found that something had happened to wall off the infection. When you see the loose tissue in the cadaver you would think nothing could stop it.

MEETING OF THE SOUTHERN SECTION HELD IN NORFOLK, VIRGINIA, JANUARY 19, 1935, UNDER THE CHAIRMANSHIP OF DR. BEVERLEY R. KENNON, OF NORFOLK, VIRGINIA.

CLINICAL BIOCHEMISTRY IN THE TREATMENT OF EAR, NOSE AND THROAT DISEASES

By F. B. BLACKMAR, M.D.

Columbus, Ga.

In a previous paper<sup>1</sup> a plan was attempted for connecting some of the factors being studied with present conceptions of allergy and infection. When the paper was finished it was seen that only a few paragraphs at the end made any attempt toward the application of these ideas to the otorhinologist's daily work. The present paper is an attempt to give more time to the application of these ideas to patients.

Nutritional chemistry, in relation to cattle and fertilizers, has made great advances. No one thinks it remarkable for a planter to supply extra calcium when he limes his soil for alfalfa, nor when he supplies potash, phosphorus or nitrogen. Contrast the above factors, calcium, phosphorus, nitrogen and potash, which are studied and considered all important by the planter, with our old idea of carbohydrate, fat, and protein, which gives no consideration to these elemental and prime factors. Individual tissue changes suggest a need for some elements and an over supply of others. Many diseases and many ordinary symptoms are really cries for something to relieve a deficiency, or to balance an excess. The newer attitude is to consider a person's intake of calcium, phosphorus, iron, iodine, sulphur, nitrogen, sodium, and potassium; and where a deficiency is suspected to attempt to supply the need by changes in diet or by the administration of medicines. This is not so difficult as it might seem. It is not necessary to memorize long tables of chemical analyses of foods. One's general knowledge with a little help will often be found quite sufficient. There are not a great many *classes* of foods and the outstanding chemical characteristics of each are not difficult to keep in mind. In selecting drugs this is a new point of view and often drugs formerly used but rarely seem to gain in importance. In some instances manufacturing houses have cooperated by evolving entirely new agents and in one instance (the use of suprarenal cortex



extract) it was found best for several members of the group to freshly manufacture their own glandular extracts.

Of the three major groups of factors, (1) autonomic, (2) glandular, and (3) chemical, it is impossible to say which is most important. The autonomic system largely regulates the activities of the glands of internal secretion. It is not unreasonable from many findings to believe that the chemical equilibria also produce part of their effects through stimulation or inhibition of the anabolic or catabolic glands of internal secretion and so each of the three groups of factors are interlocked with each other to form a chain.

As the studies of the Jarvis correspondence group progressed it became evident that many diseases are promoted by one group of nutritional factors and healing is promoted by their antagonists. Thus the diseases and etiological factors of disease, according to present conceptions of this group of observers, are placed in two antagonistic groups.

Certain of the factors belonging to opposite main groups seem to pair off as special antagonists. There are acids combating alkalies, sodium combating potassium, calcium versus phosphorus, sulphur and light metals versus the heavy metals, and the catabolic glands of internal secretion versus the anabolic glands.

The resultant of these forces may be visualized as a wagon with many horses attached on each end pulling in opposite directions. As a result of this tug of war the wagon moves forward and backward. Various tissue changes accompany displacement in one or the other direction.

Changes in the color of the nasal septum give those who have trained themselves to judge these colors the most easily obtained *preliminary* idea of the position of the wagon in the above illustration, subject to the following limitations.

The author personally feels that the color of the nasal septum indicates the *local* resultant of the antagonistic factors of the autonomic nervous system; the resultant of the antagonistic secretions of the internal glands and the resultant of chemical antagonisms in the intercellular fluid in the nasal mucosa; and *generally but not always* the resultant of *these factors* in the body as a whole. Occasionally patients are seen where the history, basal metabolism, urinary acidity, blood pressure and therapeutic results do not confirm the indications of the color of the septum. This is usually a local displacement in the septal color not in accord with the resultant in the body as a whole and in most instances the local factor can be found. If the sinus infection, dental abscess, or whatever it is, is corrected

then frequently the septal color will truly indicate the resultant of the body as a whole.

A factor in the septal color change can be the actual color of the blood itself. From anemia there is a tendency toward paleness of the septum. However, even with anemia there *may be* dilatation of the vessels from changes in nerve stimulation and the septum may present various shades of redness despite the anemia. In the interpretation of color changes of the septum the hemoglobin concentration can be determined and any anemia which might be present given due weight.

The author's present conception is that the best clinical biochemical approach to a patient's problem is to familiarize oneself with the clinical effects of chemical, autonomic and glandular deficiencies and excesses. Armed with this information the question in histories will naturally yield more information pertinent to the patient's symptoms and tissue changes, and corrective agents will suggest themselves.

#### THE AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is composed of two antagonistic parts; the sympathetic and the parasympathetic systems. The latter is the cranial and sacral part of the system. There is a constant attempt between these two parts to maintain a balance. Frequently this balance is slightly upset for short periods of time, but it should soon return to a condition of equilibrium. Following exercise fatigue, fear, anger and under the influence of certain drugs the sympathetic system easily dominates the control of the body. During such periods the reserves of the body are mobilized and depleted. Animal starch, called glycogen, changes to blood sugar during the period of sympathetic dominance. A stored substance in the cortex of the suprarenal gland is also mobilized, resulting in a loss in weight in these glands. At other times, such as during rest, the parasympathetic system dominates the body. Reserves are again built up. Blood sugar is removed from the blood and stored as glycogen during this period.

The sympathetic system is composed of a network of connected nerves. An impulse at one point, if strong enough, can travel throughout the entire system. The effect of these impulses varies according to the part affected. Thus the abdominal vessels contract, forcing more blood away from the organ of storage (the liver and spleen) to the skeletal muscles, heart, lungs, brain and, perhaps, the *nasal septum*.

Such facts as the following lead to the belief that sympathetic activity, besides resulting in the well-known vascular dilatation of

the brain, heart and lungs, is also regularly associated with vascular dilatation in the nose and a red nasal septum. In a number of patients with pathology near the thoracic or cervical sympathetic chain, on one side only, it has been seen that the septum on this side was more red than on the other side. When a red septum is seen on one side only the author considers the possibility of a local staphylococcus or streptococcus infection in the sinuses, then looks for abscessed teeth, especially an upper tooth on the red side, then at the tonsils and cervical lymph nodes on the red side, then for chest pathology on the red side, then for an appendix or even a tube on this side. This idea of sympathetic irritation is not at all new, for years ago a series of papers suggested a search on the side of a slightly enlarged pupil and a single drop of homatropin was instilled into each eye, noting the effect on the two pupils. Sympathetic irritation was suspected on the side upon which the pupil dilated most rapidly. In the case of a quiescent appendicitis the author has seen a bilateral redness of the septum which could not be explained until the appendix became more active. Following the appendectomy the septum returned to its normal color.

The word edema as used in connection with cardiorenal disease and edema as used in relation to the nasal mucosa in allergy are not only different, but clinically it appears that the factors which benefit one are harmful to the other. No satisfactory explanation has been found in the literature available and as it was considered a matter of fundamental importance the author has indulged in the following theoretical explanation.

What tissue changes are at the bottom of changes in the color of the nasal septum? Three primary changes may be considered: first, changes in the size of the capillaries; second, changes in the volume of the septal tissue cells; and third, changes in the amount of fluid around and between the septal tissue cells. Changes in the size of the capillaries are generally accepted as being due to changes in dominance between the local sympathetic and parasympathetic nerve supply to these capillaries. Unless there is a purely local force acting, the effects of the varying dominance of the two parts of the autonomic nerve supply in the nasal septum are thought to reflect the resultant of the body equilibrium and are paralleled by the effects of the secretions of the anabolic or catabolic glands of internal secretion. The catabolic secretion of the suprarenal glands, for instance, produces effects similar to that of excitation of the sympathetics. The anabolic secretion of the pancreas simulates the changes produced by stimulation of the parasympathetic nerve supply to the

pancreas. The amount of fluid outside the vessels and in between the cells is largely controlled by osmosis and the permeability of the vessel walls. The volume of the individual septal tissue cells varies, because of osmosis, with alterations in the chemical constitution of the fluid surrounding the cells.

The fluid around the body cells, according to such formulas as that of Ringer's solution, which are attempts to duplicate the mineral composition of the blood plasma, contains about eighteen times as much sodium chlorid as potassium chlorid. On the other hand, the chemical composition of the entire body reveals twice as much potassium as sodium. It would seem that roughly the cells of the body must contain mostly potassium in contrast to sodium in the plasma and intercellular fluids. There is said to be a fatal dose of potassium in the red blood cells alone if it were free in plasma. The cell wall in a way is a semipermeable membrane separating potassium ions inside from sodium ions outside. When sodium is absorbed and ionized it adds to the ions outside the cell and, since water passes from the lighter to the more dense side of the cell wall, there is exosmosis and the cell becomes smaller, and if it is a secretory cell it secretes less. When potassium is absorbed the great majority of it certainly leaves the plasma and passes into the cell, adding to the density within the cell. As a result fluid, to dilute the incidental concentration, passes in, following the potassium. The volume of the cell increases and its secretion increases. Another effect of increase in the size of the cells is pressure upon the capillaries. The increase in cell volume at the expense of the blood vessels tends to cause a paleness in color. If the cells continue to swell, the nerve endings are irritated by pressure and sneezing and other allergic sensations result.

Apparently the same changes take place in the kidney as in the nasal secretory cells and with the administration of potassium there is diuresis corresponding to nasal rhinorrhea. With increases in the intake of sodium there is a decrease in urinary secretion. The analogy between the nose and kidney is widened by the observation that a common prodromal symptom of an allergic attack is polyurea.<sup>2</sup>

Nasal edema occurs in association with those factors on the side of parasympathetic dominance and is the opposite condition to the vascular congestion which occurs with those factors on the side of sympathetic dominance.

Sodium chlorid and a meat diet are ill advised in cardiorenal edema, but are often helpful in nasal allergic edema. Acid is used



as a diuretic in some cases of nephritis which would seem quite contrary to the above theory. It may be that these are allergic kidneys in which cellular enlargement is relieved as a result of acid administration and the circulation is improved, resulting in diuresis. If this is true such patients, in whom diuresis is produced by the use of dilute hydrochloric acid, might be expected to show pale septa rather than the red septa usually seen in nephritis.

In reference to the patient's psychology the color of the septum reveals very definite pictures. A red septum patient often awakes in the morning unrefreshed and still fatigued. He is conscious of muscular strength and may be best described as a fatigued strong person. Cannon<sup>3</sup> has shown that fatiguing emotions produce bodily changes which fit in with this conception. The pale septum patient, always granting that the color of the septum reflects the general bodily condition and is not a purely local alteration, is usually more phlegmatic and feels weak. Red septum patients need all the rest they can get while pale septum patients should be encouraged to attempt exercise, stopping upon the first signs of fatigue, but trying again after short rests.

The difficulty is that red septum patients who need rest are apt to be restless intensive workers, while the pale septum people who need progressive exercise usually feel so weak they are hard to stir into activity. There are many exceptions to this conception as, for instance, several doctors among the author's friends, who are noted for their untiring ability and yet have pale septa and a low blood-pressure and are mildly allergic. Pale septum patients frequently state that they have difficulty in keeping warm and their fingers and toes go to sleep and feel numb when they lie down or sit still for a while.

The blood-pressure is of great help in understanding patients' psychology from the point of view of the resultant of the various equilibria or, let us say, the septal color. A young adult with a red septum will probably have a normal blood-pressure, unless there is advanced nephritis or diabetes with vascular sclerosis, when it would be elevated. A red septum and low blood-pressure are contrary indications which call for additional study of the case. Often a local cause for the red septum will be found and, if corrected, the septum will correctly indicate the patient's autonomic, chemical and glandular resultant as being displaced toward the side of parasympathetic dominance. Low systolic blood-pressure would be expected with a shift of the body resultant (or the autonomic level) toward the pale side. With the administration of suprarenal extract prepared

for oral administration this low blood-pressure soon rises to normal and often remains there long after its administration has been discontinued. It does not rise above 120 usually, even if the extract is continued. In working with this extract it commonly happened that pale septum patients were often seen with a blood-pressure as high as 240. The oral suprarenal extract was given for relief of nasal complaints with fear and trembling in tiny doses, for in many cases proof that it elevated low blood-pressure was at hand. In place of rising, such pressures gradually fell, often to as low as 120. In old people the finding of pale septa and high blood-pressure is a most common occurrence. The improvement in these patients' general feeling is indeed gratifying.

#### GLANDULAR EQUILIBRIUM

A red septum from a glandular point of view directs our attention to the possibility of over-active suprarenals, thyroids, posterior pituitary, gonads, or a weakened pancreas. The basal metabolism may direct our attention to the thyroid as the over-active gland or the blood-pressure and urine acidity may direct us to the pituitary and suprarenal glands, respectively, but, with the exception of insulin for well-developed pancreatic weakness, dependence is generally placed upon attempting to vary the chemical equilibrium as a means of correcting the patient's imbalance, thus freeing him from a predisposition to staphylococcic and streptococcic infection, his abnormal mental irritability and his feeling of fatigue. A pale septum indicates an excess of insulin or an uncompensated deficiency in the suprarenals, thyroid, posterior pituitary, or gonads. Attempts toward correcting the imbalance consists mainly in the administration of properly selected chemicals as drugs or dietary supplements, and avoiding rapidly absorbed carbohydrates which are thought to add to the overstimulation of the pancreas. *Small* doses of insulin at from daily to weekly intervals have been used in certain types of cases to stimulate the antagonists of the pancreas, the suprarenals, thyroid and pituitary glands. This seems to have proven itself a therapeutic procedure of definite value in a variety of disturbances associated with pale septum displacements.

#### THE EFFECT OF MENSTRUATION ON THE COLOR OF THE NASAL SEPTUM

In several instances an error in estimating the autonomic level has been made due to impending menstruation. For a day or two before the commencement of menstruation there is often a temporary

shift toward redness. This will partially balance a pale septum, a neutral septum may become red or slight imbalance to the red side will become exaggerated. With the commencement of menstruation the color of the septum returns to its usual color plus a slight shift to the pale side. The natural inference would be that this paleness is due to loss of blood, but from an autonomic point of view it may be evidence of an autonomic shift or both factors may share in producing the result. Although it has not so far been the author's good fortune to examine the color of the nasal septum in a patient with menstrual epistaxis (vicarious menstruation) it seems reasonable to associate it with this shift toward active congestion in the nose.

### CHEMICAL EQUILIBRIA

Disturbances of the acid base ingestion ratio probably assume first place in the chemical equilibrium. An excessive intake of non-oxidizable acids or acid-ash foods on one hand, or alkaline foods on the other, often seems to be alone responsible for many patients' imbalances, since disappearance follows the indicated corrections. According to the doctor's inclination he can rest with asking how much meat, starches, sweets, fruits and vegetables are eaten or he can ask the patient to keep a diet history, until the next visit. The latter method *must* be used at once if the patient is recognized as one of the many who anticipate criticism and deny excesses. They must receive no suggestion of criticism until a record of a few days is at hand which cannot be denied. In general, meats, fats, fish, and carbohydrates can be considered acid foods tending to produce a red septum and its incidental pathological manifestations or to relieve the pathologic manifestations connected with a shift toward a pale septum. Almost all fruits and vegetables are alkaline foods tending to relieve symptoms associated with red septa.

Hydrochloric and nitrohydrochloric acid or sodium acid phosphate are the principal agents of this group. It is probable that these agents should not be continued after the urine acidity is elevated moderately or the reaction of the saliva becomes acid. Should this occur other agents can be used in attempting to produce a resultant shift toward sympathetic dominance. As the absorption of both sodium and phosphorus aids in shifting from a pale septum displacement it would seem that the use of sodium acid phosphate might have a triple indication.

Pale septum patients, besides the allergic complaints which bring them to the rhinologist, often will give a history of digestive dis-

orders of various types. It is believed by some internists that gastric ulcer in these patients is of an allergic nature. At any rate low or absent hydrochloric acid in the gastric secretion would furnish a double indication for the administration of dilute hydrochloric acid or nitrohydrochloric acid. Many of these pale septum patients complain of choking fullness in their throats. Many have learned to relieve this by taking soda which quickly empties the gastric content. But it is well to remember that free hydrochloric acid in excess of that which can be absorbed by the food in the stomach is the natural means of relaxing the pyloric sphincter and so a sufficient dose of hydrochloric acid may produce the same result eventually as is so rapidly accomplished by the use of soda.

All of the organic acids except oxalic form salts which are absorbed and with the elimination of carbon dioxide the alkaline ions remain. After absorption citrus fruit juices, berries and fruits which contain organic acids really tend to increase the alkaline reserve of the body.

With red septa potassium bicarbonate and citrate have each a double indication since potassium tends to produce a shift away from a red septum and the bicarbonate and citrate ions are efficient alkaline agents.

The clinical application of knowledge in reference to the sodium potassium equilibrium is that salty foods should be withheld from red septum patients and they should be encouraged to eat more leafy vegetables which are especially rich in potassium. There is on the market a physiologically balanced salt which closely resembles the composition of human plasma. It is claimed that since its sodium is balanced with calcium and potassium it does not increase any sodium excess already present and that patients do just as well with food flavored with it as on a salt-free diet. The author has found it quite impossible to tell this salt from sodium chlorid by looks or taste. Where active medication is needed, besides reducing the salt intake, to balance an excess already present, the same firm offers tablets with the same formula as the table salt except that *all* sodium is eliminated. These tablets practically serve the same purpose as alkaline medication.

An excessive intake of salt is interesting in view of the stimulating effect of salt upon the suprarenal glands. Dogs deprived of their suprarenal glands live longer if quantities of sodium chlorid are given. On the other hand, patients with deficient suprarenals are made worse by a salt-poor diet and this salt-poor diet has been used as a provocative test for Addison's disease.<sup>4</sup>



In the selection of an alkaline agent it would seem that the old A. B. C. mixture would be hard to improve upon as it consists of the acetate, bicarbonate and citrate salts of potassium, thus tending to produce a shift toward the pale side with both ions of each ingredient. Sodium bicarbonate would seem less efficient due to sodium being a factor exerting a force toward sympathetic dominance. If this A. B. C. mixture is given after meals a slow absorption is provided. When given on an empty stomach even red septum patients often notice a tingling of the extremities such as pale septum people often experience constantly.

Sodium chlorid would seem to be an ideal agent for attempting a shift toward the red side as both the sodium and chlorin ions are thought to exert force toward sympathetic dominance.

Intravenous hydrochloric acid has received much notice recently and the author prefers not to praise or condemn its use, but only to state that patients with pale septa have been seen to rapidly shift in color toward redness following a series of such injections. It seemed to him in many of these patients that improvement in their allergic symptoms accompanied the shift in the color of the nasal septum.

The fluid tolerance of an individual probably depends largely upon the sodium potassium equilibrium and patients with red septa seem to improve with the use of fluids in large quantities. On the other side, the author has seen allergic symptoms entirely relieved after the deliberate excessive use of water by patients had been discovered and stopped.

Calcium and phosphorus constitute an important equilibrium in the body. Phosphates exert force upon the total resultant toward the production of sympathetic dominance, using this term to include the resultant of all factors. Since the great majority of allergic patients have pale septa, indicating that their autonomic levels have been displaced toward parasympathetic dominance, phosphates are often more important in the relief of allergy than calcium. A certain amount of ionized calcium is necessary to prevent excessive capillary permeability, but past this point calcium actually becomes harmful to pale septum people exaggerating their muscular weakness and numbness of peripheral areas of the body. Phosphates, on the other hand, tend to produce a feeling of strength. Meats are high in phosphates and supply an excellent source. In milk the phosphates are largely balanced by calcium as calcium phosphate. Red septum patients are apt to need calcium and its absorption should be promoted by the concurrent use of vitamin D. Since vitamin D aids in the absorption of both calcium and phosphates it is apparently indicated

for both the red and pale septum patient. Newly formed cavities in the teeth indicate calcium poverty.

Although chlorids have been accepted as factors exerting force toward sympathetic dominance by all members of the Jarvis group, iodids are thought by the majority to be indicated with a red septum. The author partially disagrees with them and feels that excellent results can be obtained in some asthmatics by iodized oil injections into the trachea. Iodin vapor in the nose often helps hyperesthetic rhinitis with a pale septum and iodine powder is of definite value in chronic otitis where local vascular sclerosis has replaced active congestion. In the author's experience acute otitis media is made worse by the use of iodine powder. Subject to correction he believes that the entire halogen group should be considered as exerting force toward the production of sympathetic dominance.

Iron, copper, manganese, mercury and arsenic, aside from the effect of some of them on the correction of anemia, are thought to be indicated to correct parasympathetic dominance. In many ways sulphur can be pared off as their antagonist. It may be that the high sulphur content of eggs and onions may have some part in the frequent allergic symptoms following their absorption. If used at all, colloidal sulphur should be given with care to those who have parasympathetic dominance, otherwise distressing reactions will follow either its oral or intravenous use. As calcium seems to exert force toward the correction of sympathetic dominance, calcium thiosulphate would seem preferable to sodium thiosulphate for those with sympathetic dominance caused by an excess of the heavy metals.

In connection with arsenic as a force toward the production of sympathetic dominance, it should be noted that an excessive sodium intake predisposes to arsenical dermatitis and treatment should include such recognized sodium antagonists as potassium, calcium and magnesium.

If the vasodilatation accompanying certain atmospheric changes has been thought to have a beneficial effect upon syphilis, surely the same result when produced by arsenic or mercury would be likewise beneficial if they are used in sufficient doses to produce and maintain a shift toward sympathetic dominance.

Magnesium and sulphur are of benefit to red septum patients and milk of magnesia after meals would probably have to be taken in large quantities by those with sympathetic dominance to produce purgation, but any part which might be absorbed would have a corrective influence. Magnesium citrate might be selected for those with sympathetic dominance with a similar thought in mind.

Strychnin is a drug which is indicated in some pale septum patients. But to the author the selection of the patients who should receive it seems difficult. The activating sparks to the adrenals, thyroid and probably other catabolic glands come through their sympathetic nerve supply and in patients without exhausted catabolic glands the use of strychnin, which increases the sympathetic impulses, is certainly of great benefit, at least temporarily, while other slower factors are being built up. But with pale septum, parasympathetic dominant patients, due to exhaustion or constitutionally inferior catabolic glands, the effect of added sympathetic impulses is to prevent these glands from accumulating the reserve without which each emotional excess results in general prostration. A history of exacerbations of symptoms following emotional stress seems to the author to constitute a contraindication to the use of strychnin and an indication for the administration of a suprarenal extract prepared for oral administration in the hope of relieving the drain upon the suprarenals until they can accumulate a reserve. If they are constitutionally inadequate (an extreme example being Addison's disease) continued administration will be necessary as with insulin in diabetes.

Tablets containing atropin and quinin seem to help pale septum patients, atropin being one of our most potent sympathetic stimulants. Quinin would seem useless in the amounts contained in the usual rhinitis tablets, but the small quantity used does seem to be potent and theoretically correct. The suprarenal glands are particularly absorbent in reference to quinin and large doses result in suprarenal depression. Small doses, however, result in stimulation. Very small quantities must be used in pale septum people to obtain suprarenal stimulation, care being taken to avoid suprarenal depression which would, of course, defeat the aim of the treatment since pale septum patients are the patients who benefit most from the use of these tablets. Incidentally it would seem advisable to highly fortify quinin with suprarenal stimulants when giving it for preventing or treating plasmodial infections. Alcohol in sufficient quantities to temporarily exhaust the pancreas is probably a good plan. Naturally a small quantity of alcohol might overstimulate the pancreas, resulting in hyperinsulinism, which would still further submerge the depressed suprarenals.

Large quantities of quinin can be used in red septum patients in an attempt to relieve a "congested headache."

Large quantities of pepper and alcohol seem to favor a shift to the red side of sympathetic dominance and it is interesting to note the tendency toward a lower average basal metabolism in the tropics

and the free use of pepper and alcohol by the white races inhabiting such lands. Whether the heat or an excessive fruit and vegetable diet is responsible or both is a matter for speculation. Heat and humidity have been described as a suprarenal depressor to the point of death from diarrhea in the Far East<sup>5</sup> and, on the other hand, fruit and vegetables are almost certain to supply an alkaline diet. Doctor Jarvis reports that most of his patients in Vermont have red septa, while those members of the group who live in the South report that most of their patients the year round show pale septa. If this observation is true, excellent medical therapy for acute rhinitis, let us say for instance in the East, may not be best in the South.

Nicotin from the glandular point of view seems to parallel quinin in its effect, small quantities stimulating the catabolic glands and large quantities depressing them. If it is remembered that increased blood sugar follows the eating of sweets and likewise the stimulation of the catabolic group of glands the reason for the arrest of hunger after a meal by smoking is apparent. The effect of an after-dinner smoke would seem to be somewhat similar to that of a sweet desert. Excessive smoking is probably harmful to pale septum patients with catabolic glandular insufficiency.

Peptone was reported in 1922<sup>6</sup> as causing an enormous increase in the salivary response to the parasympathetic stimulating drug pilocarpin. The excitability of the vagus part of the parasympathetic system was shown in 1928<sup>7</sup> to be increased by the injection of foreign serum into an animal. In 1930<sup>8</sup> peptone injections were shown to increase the reactivity of the heart to pilocarpin.

The above researches would suggest that peptone and foreign serum are powerful parasympathetic stimulants and should be of great value to red septum, sympathetic dominant patients. Clinically this seem entirely true. It is hard to believe that any one has failed to see the harmful effects of foreign proteins in allergic patients, presumably from stimulation of their already dominant parasympathetic system.

It has been customary to consider all foreign proteins as having identical effects. It seems to the author that this belief is subject to doubt as a number of diseases in his practice have been seen to result in pale septa and there are facts which suggest that other diseases may fall on the side of parasympathetic dominance. In his practice the clinical picture called influenza is associated almost invariably with a pale septum. Another member of the group who lives in the northeastern United States reported that his influenza patients (presenting the same clinical picture) showed red septa in most instances.



In closing, the following tentative grouping of diseases as to the apparent effects of their toxins is offered. Staphylococcic infections and streptococcic infections, up to a certain degree of severity, seem to tend to produce sympathetic dominance. Very severe streptococcic infection may produce signs of parasympathetic dominance such as urticaria, probably by suprarenal exhaustion. Atropic arthritis is said to be associated with, and promoted by, sympathetic dominance.<sup>9</sup>

Migraine and epilepsy are frequently associated with pale septa parasympathetic dominance and hyperinsulinism or hypoadrenalism.

Intestinal parasites not only are frequently associated with pale septa and parasympathetic dominance, but there is often an increase in eosinophiles as in allergy.

Diphtheria and Vincent's angina are probably diseases associated with parasympathetic dominance and pale septa. In connection with diphtheria, probably a pale septum disease, it is interesting to note that a persistent acid reaction interferes with the production of toxins<sup>10</sup> and if sugar is present in the broth in considerable quantity the acid produced by its fermentation hinders materially the accumulation of toxins. Broths free of muscle sugar are advantageous. Before the days of antitoxin, clinicians<sup>11</sup> pinned their therapeutic faith to quantities of iron (now believed to be a red septum producing factor).

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## DIAGNOSIS OF ACUTE SUPPURATION OF THE PETROUS PYRAMID

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Suppuration of the petrous pyramid is the most recently recognized complication of purulent otitis media. It is caused by extension of the infection from the middle ear or mastoid by way of the cell channels leading into the petrous pyramid.

Previous to 1929, very few cases were diagnosed before autopsy. During the period from 1929 to 1931, Kopetzky and Almour,<sup>1</sup> Eagleton,<sup>2</sup> Profant,<sup>3</sup> Friesner and Druss,<sup>4</sup> and Voss<sup>5</sup> were among the first to formulate a clinical picture of this condition. Ramadier<sup>6</sup> has published the most extensive treatise appearing since 1931. Credit for the stimulation of this work is due to those men who studied the causes of sixth nerve paralysis. Among those who contributed much may be mentioned Dorello<sup>7</sup> in 1906, Wheeler<sup>8</sup> in 1918, Vail<sup>9</sup> in 1923 and Sears<sup>10</sup> in 1925.

At autopsy on patients dying from otitic meningitis, careful scrutiny frequently revealed necrotic areas on the surface of the petrous pyramid, thus emphasizing the urgent need for diagnosis of the pre-existing suppuration.

The many discussions in the recent literature establish the fact that Gradenigo's syndrome, consisting of paralysis of the external rectus muscle and unilateral temporoparietal pains in the presence of acute otitis media of the affected side does not typify a clinical entity, and should not be confused with the picture presented by petrous suppuration. It is the belief of Kopetzky,<sup>11</sup> Eagleton<sup>12</sup> and a large number of other prominent otologists that Gradenigo's triad of symptoms is often the result of extension of the infection by way of the veins—retrograde thrombophlebitis, or is toxic in origin, factors entirely outside the petrous pyramid, causing a mild localized area of meningitis involving the dura in contact with the Gasserian ganglion and sixth nerve. Supporting this opinion is the fact that unilateral temporoparietal pain and sixth nerve paralysis appear as early symptoms, sometimes before suppuration in the mastoid is established, and in rapid sequence, whereas if abducent paralysis does occur with empyema of the petrous pyramid, it is usually a later symptom, appearing when the infection has reached the tip cells of the pyramid.

## ANATOMY

The following brief consideration of the anatomic relations associated with the symptomatology will help clarify its interpretation.

*Bone.*—The acute causes of suppuration of the petrous pyramid occur most frequently in pneumatized mastoid bones. Extensively pneumatized temporal bones present cellular development extending

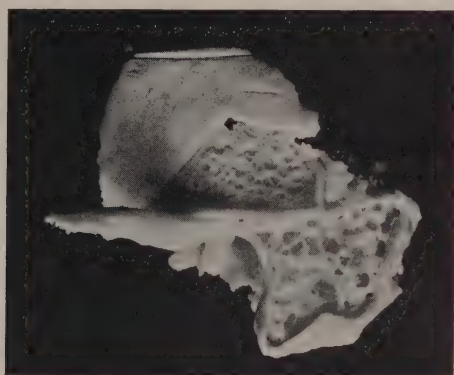


FIG. 1. Temporal bone with pneumatization of zygoma and squama. From collection of E. B. Burchell.

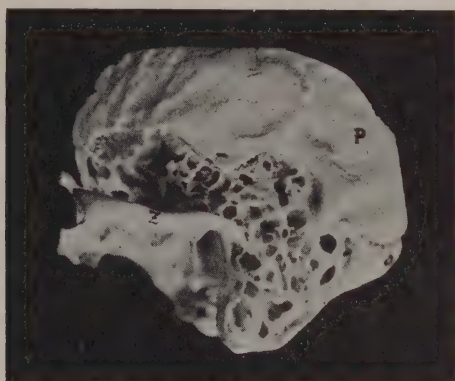


FIG. 2. Extensive pneumatization of temporal bone. From collection of E. B. Burchell.

into the zygoma, the squama, the occipital bone, the floor of the middle ear, around the mouth of the eustachian canal, well down into the anterior wall of the mastoid tip and into the petrosal pyramid. (Figs. 1, 2, 3, 4, 5.)

Carmack,<sup>13</sup> in a dissection study of ninety-four petrous pyramids in adults, found 32 percent pneumatized. He states, "There is every anatomic reason for extension of an inflammatory process into the

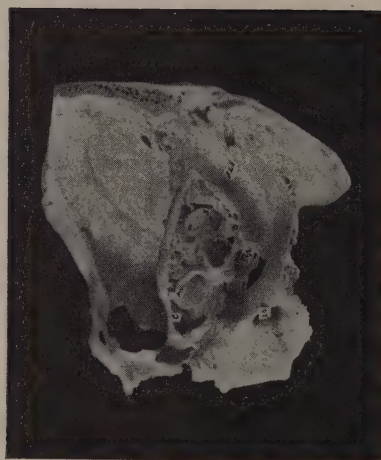


FIG. 3. Pneumatization of the petrous pyramid.  
From collection of E. B. Burchell.

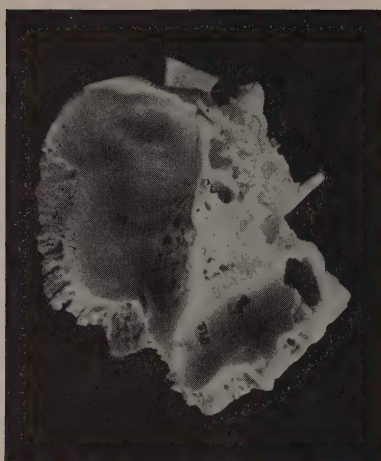


FIG. 4. Large cell in the apex of the petrous pyramid.  
From collection of E. B. Burchell.



FIG. 5. Pneumatized temporal bone with large cell in apex. From collection of E. B. Burchell.



cells of the pyramid, with otitis media, similar to that occurring in a mastoid process."

Hagens<sup>14</sup> studied fifty temporal bones and found 34 percent had cell spaces in the petrous pyramid.

Myerson, Rubin and Gilbert<sup>15</sup> examined 200 temporal bones, both before and after removal from the cadavers and, by gross section, found only 11 percent pneumatized petrous tips. This low percentage is largely due to the twenty-three specimens from infants. An interesting finding in their series is that fifty-four temporal bones from negroes showed three times the percentage of cellular petrous pyramid as did those in white subjects.

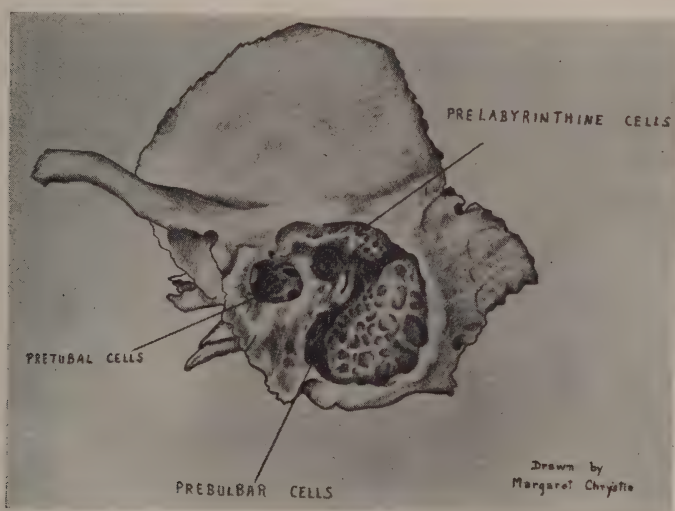


FIG. 6. Areas of entrance of cell pathways.

Glick<sup>16</sup> states that "pneumatic cells are present in the apex of the human temporal bone more frequently than is generally believed." In a microscopic study of the petrous apex, he found that inflammatory changes extended with less proliferative activity in the marrow than in the lining membrane of the cells.

Eagleton<sup>17</sup> has found infection extending by way of marrow-filled cancellous tissue into the petrous apex.

*Pathways.*—The most generally recognized cellular pathways leading into the petrous pyramid are those described by Kopetzky.<sup>18</sup> (Fig. 6.)

1. From the anterior or tympanic space above or behind the superior semicircular canal, following the posterosuperior surface of the petrous pyramid into the tip.

2. From the peritubal cells into the pyramid.
3. From the peritubal cells by way of the carotid canal into the tip.

Ramadier,<sup>27</sup> in an extensive treatise in 1933, pictures the entrance of six avenues of infection as follows: (1) Prebulbar, (2) pretubal, (3) sublabyrinthine, (4) postlabyrinthine and (5) supralabyrinthine.

*Nerves.*—The great superficial petrosal nerve, which in all probability carries sensory as well as motor fibers, according to Piersol,<sup>19</sup> arises from the facial nerve in its canal, comes to the surface of the petrous pyramid through the hiatus fallopii, passes usually in a groove on the surface of the petrous pyramid outside of the dura, for a distance of 8 to 10 mm., before disappearing under the Gasserian ganglion to reach the foramen lacerum, where it joins the great deep petrosal nerve to form the Vidian nerve, thence through the Vidian canal in the sphenoid bone, the ascending orbital branch passing through the sphenomaxillary foramen to supply the periosteum of the orbit, the posterior ethmoid cells, etc.

Vail,<sup>20</sup> speaking of great superficial petrosal nerve neuralgias, says: "This neuralgia is of extreme diagnostic importance when we are dealing with a case of suppurative otitis, because one should look upon the complaint of pain around the eye, back of the eye, in the temporal region and back of the head as pathognomonic of irritation somewhere in the course of the great superficial petrosal nerve and that place can only be in the petrous apex."

The trigeminus or fifth cranial nerve reaches the surface of the petrous bone through an opening in the dura after leaving the lateral side of the pons. The sensory roots form the Gasserian ganglion which is enclosed in Meckel's space, a delamination of dura lying in a slight depression on the apex of the petrous bone; internally, the Gasserian ganglion comes in relation with the cavernous sinus and internal carotid artery. Of the three branches given off from the Gasserian ganglion, we are concerned only with the first or ophthalmic branch. This branch is more firmly bound down by the dura in its longer course within Meckel's space than either of the other two, subjecting it to greater tension when irritation of the Gasserian ganglion occurs; the ophthalmic nerve passes upward and forward in the external wall of the cavernous sinus for about 25 mm., thence through the sphenoid fissure to supply the lacrimal gland, the upper eyelid and the skin around the external canthus.

*Abducent or Sixth Nerve.*—This nerve is exclusively motor in function, supplying the external rectus muscle of the eye. Leaving the lower surface of the pons, it pierces the dura over the sphenoid

bone, turns forward and passes between the apex of the petrous bone and posterior clinoid process of the sphenoid bone. A strong, fibrous bundle connecting these two bony prominences forms Dorello's canal. The superficial petrosal sinus as well as the abducent nerve passes through the canal. Passing forward and upward to the orbit, the abducens supplies the external rectus muscle.

#### SYMPTOMATOLOGY

*Aural Discharge.*—The discharge from the ear is continuous from the onset of the otitis or reappears in from two to six weeks following mastoidectomy. In forty-eight cases collected from the literature, where the facts are noted it was continuous in thirty-three cases and reappeared in fifteen. In three cases of my own, two were acute with continuous discharge, and one, a chronic case, had intermittent discharge over a period of four years; was then quiescent for six years, until death from meningitis revealed, at autopsy, necrosis of the petrous apex. Many of these cases of continuous or reappearing discharge are reoperated upon one or more times, with the hope of locating an overlooked focus in the mastoid, only to discover eventually that the discharge is finding its way through a fistulous opening from the petrous bone into the middle ear or mastoid.

The bacteriology is not significant.

The continuance or reappearance of a profuse discharge, especially when accompanied by temperature and pains around the eye of the same side, two to six weeks after mastoidectomy, points strongly towards petrous pyramid suppuration.

*Pain.*—This is the most characteristic and constant symptom. It is located in back of, and around the eye, in the temporoparietal region, sometimes reflected to the occiput of the affected side. At first it is usually nocturnal in character, described as a severe, agonizing, deep pain in the eye, with a feeling of the eye's being pressed forward. As the disease progresses, the periods of severe pain may change, occurring during the day, the severity of the pain lasting a few hours at a time. If questioned carefully, the patient is found rarely to be entirely free from pain in the parietal region.

Between the attacks of severe pain the patient is often fairly comfortable. If seen in the afternoon following a nocturnal attack it is hard to realize the extent of the suffering described by the patient, and he is put down as a neurotic. A note charted by the interne on a patient whose condition later was diagnosed as petrous pyramid suppuration, reads: "She is about the worst neurotic I have ever

seen." This nervous irritability is more or less characteristic of the disease.

This peculiar type of eye and associated pain persists and becomes more intensified until free drainage is afforded either by surgical means, by a large fistulous opening formed by necrosis into the middle ear or rupture of the cortical surface of the petrous apex. If the latter occurs, the patient goes into the quiescent stage and soon develops the terminal stage of meningitis.

Kopetzky<sup>21</sup> states of this peculiar eye pain: "It is highly significant of petrous pyramid suppuration," and explains it as the result of an irritation of the ophthalmic branch of the trigeminus where it is firmly bound down in its course from the Gasserian ganglion to and through the cavernous sinus.

Vail<sup>22</sup> writes: "The eye and orbital pain in cases of suppuration of the apex of the petrous bone is not due to the irritation of the Gasserian ganglion, but to the greater superficial petrosal nerve."

Eagleton,<sup>23</sup> discussing Vail's paper, is unable to say from his cases which nerve causes pain; probably both the fifth and the Vidian nerves are responsible in different cases, depending upon the position of the lesion and type of bone.

Owing to the position of the great superficial petrosal nerve in a groove in the petrosal cortex, outside the dura, and 10 mm. nearer the origin of the infection, it seems logical to believe that the earlier pains come from its irritation. As the infection extends further into the apex it reaches the cells underlying the Gasserian ganglion and irritation of the ophthalmic nerve takes place. It is not so important to recognize which of the two nerves carry the pain impulses as it is to be able to interpret the cause of the pains. In many pneumatized petrous bones the cells do not extend to the apex and the irritation reflected through the thin cortex of the bone does not reach the Gasserian ganglion. If a localized leptomeningitis takes place over an infected petrous pyramid, as many authors believe, then both nerves are involved.

*Temperature* of the low grade, septic type rarely exceeds 102° F. and usually ranges from 99° to 100° F. during the day. There may be periods of several days with nearly normal temperature. The temperature remains of the above type until the terminal period of meningitis or until the suppuration is relieved by drainage.

The *blood picture* is of little value except to show that a mild infection is present, causing an increase of leucocytes from 10,000 to 15,000, the picture of a subacute infection.

The eye-grounds usually show no change.



*Roentgen Ray.*—Too much dependence should not be placed upon the roentgen ray findings. One of my recent cases of operative mastoiditis of the right side showed increased density of the left petrous bone at the first exposure.

Coates, Ersner and Myers<sup>25</sup> report nine cases of roentgenographic changes in the petrous bone, some of them without clinical manifestations. Case No. 8, with bilateral mastoidectomy, showed symptoms (Gradenigo's syndrome) on the left side, while the roentgenograph revealed changes in the petrous pyramid on the right.

Taylor,<sup>26</sup> who has worked with Kopetzky, states: "Roentgenographically the pneumatic petrous pyramid shows variations from the normal when otitic infection is present," and that "every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion in the petrous apex."

A single exposure showing the petrous pyramid is of little value. A progressive series of roentgenograms, beginning with the original mastoid exposure and showing an increasing density of the petrous pyramid of the affected side, is of great aid in arriving at a positive diagnosis.

*Transitory Symptoms.*—In the course of many of these cases symptoms of perilabyrinthitis, nystagmus, nausea and vomiting may occur. They are usually transitory, lasting only one or two days, and may be easily overlooked. They are caused by the infection passing through the cells overlying the labyrinth.

Facial weakness occurs in some cases; it is caused by an irritation of the facial nerve somewhere in its course through the petrous bone. Like the labyrinthine symptoms, it is transitory, mild in character and may pass unnoticed.

In a patient suffering from prolonged discharge following an acute otitis, accompanied by characteristic eye pain, one should always be on the alert for these less constant but significant symptoms.

*Paralysis of the external rectus muscle* is a relatively rare involvement in established cases of suppuration. It occurs only when the suppuration reaches the very tip cells of the bone involving the sixth nerve.

Kopetzky<sup>24</sup> believes that sixth nerve paralysis occurring in suppuration of the petrous pyramid is the exception rather than the rule. In two of the three cases I have had, sixth nerve paralysis occurred, but after a long period of pain, low-grade temperature and persistent discharge.

#### CASE REPORT

*Case 1.*—M. M., white, female, twenty-six years old, was first seen in consultation February 3, 1934, suffering with acute purulent otitis media on the

right side, associated with acute sinusitis of the same side and an upper respiratory infection of two weeks' standing. Temperature was 102° F. A myringotomy, followed by sero-purulent discharge, relieved the acute pain. Two days later some pain remained and tenderness developed over the antral and tip regions of the mastoid. The patient was sent to the Bryn Mawr Hospital for a roentgenogram, which revealed "a large and well-pneumatized mastoid on each side, the left clear, the right definitely hazy, and dense in the region of the antrum and mastoid tip. Definite evidence of infection with as yet no necrosis." A second roentgenogram two days later showed "an increase in density of the right mastoid, with several areas which suggest necrosis of the cell walls." This report, together with continuation of the temperature and increasing areas of tenderness and unilateral parietal headache, was sufficient evidence to warrant mastoidectomy.

At operation there was found a large area of broken down cells filled with pus occupying the space back of the antrum and in the mastoid tip. The bone was pneumatic, the cells extending into the zygoma and well down over the jugular bulb.

Except for parietal pains, which were attributed to the sinusitis, and an occasional rise of temperature to 100°, the course for the next two weeks was fairly normal. There was still some purulent discharge from the canal and mastoid wound.

During the night of the fourteenth postoperative day, severe pains in back of the eye and temple kept the patient awake in spite of large doses of morphin. The next day more pus was present in the canal and mastoid wound. The patient cried a great deal, complained of eye pain and pains in the back of the head. Roentgenogram of the mastoid showed "a few cells remaining in lower anterior portion of the mastoid and in the zygoma. No evidence of epidural abscess." Two days later mild nystagmus was noticed. It could only be seen when the eyes were turned to the extreme position. The nystagmus and some nausea lasted for three days. Examination by a neurologist was negative. Eye-grounds were negative. Two days later, February 25, the neurologist and ophthalmologist re-examined the patient, with negative findings. Blood culture was reported negative.

From February 25 to March 21 the symptoms were much less severe; the temperature was normal for periods of three to six days; the discharge continued from the wound and the canal.

March 21, five weeks after the original operation, the wound was reopened. The antral region was filled with necrotic granulation tissue, but no infected cells were found. Large areas of the dura were exposed above the antrum and over the lateral sinus but were found normal. The findings were disappointing. Suppuration of the petrous pyramid was suspected.

For two or three days following, the discharge and the severity of the pains were lessened. On the third day following the second operation paralysis of the right external rectus muscle appeared, the discharge was again profuse and the pains were increasing in intensity. A roentgenogram of the base of the skull revealed definite cellular development in the petrous pyramid of the left side. On the right, the affected side, the detail was obscured, the cell walls being barely visible. A diagnosis of suppuration of the petrous pyramid was made.

For ten days following there was a diminution of the severity of the pain, and the relatives of the patient would not consent to further operative procedure. On April 9, twenty days after the second operation, the patient had a very bad

day. Dr. F. C. Grant found no localizing evidence of brain disease. Consent for consultation with Dr. Samuel J. Kopetzky was obtained. He verified the diagnosis of suppuration of the petrous pyramid and advised operation.

The old wound was reopened and converted into a radical mastoid cavity, revealing three fistulae entering the petrous pyramid, one, with an opening 2 mm. in diameter, extending for 3 cm. into the petrous pyramid from the pretubal cells; a second, from the prebulbar cells, extending 4.5 cm. from above and in front of the superior semicircular canal; a third extended for 2 cm. into the petrous pyramid. No further operation was thought necessary for drainage. Immediately following the operation iodized oil was injected into the pretubal fistula and roentgenogram showed it to reach the petrous apex.

The pains in the eye region and the external rectus paralysis gradually lessened until there was complete disappearance in two weeks. The discharge from the canal continued in decreasing amount until October 1, 1934. Since that date the ear has been entirely dry and the patient in good health.

Bacteriologic examination revealed hemolytic streptococcus as the infecting organism throughout the course of the disease.

### SUMMARY

Suppuration of the petrous pyramid is the most recently recognized complication of purulent otitis media.

An acute case of such suppuration may resolve into a chronic purulent otitis media where there is sufficient drainage, through a fistulous opening into the middle ear or, if the cell structure is favorable, it may heal spontaneously in this manner. If drainage is not sufficient, some form of surgical assistance is necessary.

It is believed that the source of infection in many cases of chronic suppurative otitis media has its origin in the petrous pyramid.

When a radical mastoidectomy is performed for relief of chronic discharge and there is found necrotic granulation in the middle ear, especially in the region of the eustachian tube, one should suspect a fistula leading through the peritubal cell into the petrous bone.

A case typifying the symptomatology of acute suppuration of the petrous pyramid is reported.

The clinical picture of an acute suppurative petrous pyramid requiring surgical drainage is nocturnal attacks of pain in, around and back of the eye and in the temporoparietal region of the affected side, occurring with increasing intensity over a period of a few weeks, accompanying an acute purulent otitis media and mastoiditis, low-grade septic temperature and evidence of progressive involvement of the petrous pyramid, as shown by a series of roentgenograms.

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## THE EXTERNAL NOSE AS A SEAT AND SOURCE OF TROUBLE

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It is a practice among some barbarous nations to punish crime by cutting off the nose close to the face and then allowing the criminal to go free.

For the poor unfortunate it is a punishment worse than death, for he becomes an outcast among his own people, despised and shunned on all sides.

Why is it that, whereas the loss of any other organ, as the eye, for example, excites in the beholders a sentiment of pity, the loss of the nose excites disgust and repugnance.

I wonder if it is because the nose is a peculiarly human characteristic, the one feature which more than all others distinguishes man from the beast. Standing out majestically upon the front of the face, it is something very different from the snout, the muzzle or the proboscis of the lower animals.

The size and conformation of the external nose has always been regarded as of great importance in anthropologic studies. Authorities are in agreement in accepting the so-called cephalometric index, which is the ratio of the width of the nose at its base to its length, as the most valuable single physical criterion that can be found for the classification of races.

In the science of physiognomy likewise much is made of the nose as an indication of an individual's mentality or strength of character.

In general a large, well proportioned nose is regarded as an evidence of intellectuality. Napoleon, who was a good judge of character, always looked carefully at the shape of men's noses. "When I want any head work done," he once said, "I choose a man with a long nose."

Embryologically, it is a notable fact that the external nose actually develops as an outgrowth from the brain. The nasal organ first appears as small ectodermal placodes on the under surface of the fore-brain, in the region known as the nasofrontal process. Two small indentations on each side divide this process into a medial and two lateral processes. These indentations constitute the olfactory pit from which develop the nasal cavity.

At the center of the medial process there appears the so-called area of His, from which there is a downward growth which moulds itself into the formation of the dorsum and the tip of the nose.

Phrenologists perhaps will find in this anatomic fact an explanation of the nose as an index of intelligence; for must not a branch partake of the essence of its root?

As physicians we are deeply interested in the physiology as well as the pathology of the nose.

I will not go so far as to ask you to accept the theory of the external nose as an organ of ratiocination, but I do here wish seriously to protest the general tendency to regard it as a mere external appendage without useful purpose or function; or as having only the unimportant negligible function of serving as a protective covering for the organ of olfaction.

When we study the evolution of the nasal organ in the lower animals we are led to the conviction that the outer part must have developed in response to certain definite needs in connection with both respiration and olfaction.

Phylogenetically, olfaction preceded respiration as a nasal function. The external nose in its most primitive form made its appearance at the period when the latter function became grafted upon the former. In its highest development, as seen in man, it serves a purpose in properly guiding and directing the inspired air, so that it can in the most effective way subserve these combined functions.

It is now some years since it was discovered that the inspiratory air current does not take a straight course through the nasal passage but that it describes a gently upward directed curve from the anterior to the posterior nares. Paulsen, in 1882, experimenting on human cadavers, placed small strips of red litmus paper over the surface of the septum and external nasal walls, and then forced a current of ammoniated gas through the passages by means of a bellows. He found that the main current of inspired air, lifted to above the inferior turbinal, passing over the region of the middle, and then descending to the choanæ.

Francke, in 1893, using a kind of smoke on prepared models, in general confirmed the findings of Paulsen, but in addition observed that the current in its passage through the fossa regularly formed at certain points definite whorls or eddies. The object evidently is to conduct the main current in a slowed rate over the respiratory region and at the same time enable portions, by slow drift and by eddies, to come most effectively in contact with the olfactory areas. It is not difficult to see that the conformation of the external nose and the size

and position of the nostrils are essential determining factors in this regard. Downward directed nostrils tend to raise the height of the curve and slow the current; upward tilted or forward directed nostrils tend to lower its height and accelerate the current.

We are of the opinion that racial differences in the form of the external nose rest largely upon these principles. Inhabitants of dry cold climates have long noses with the nares in the horizontal plane, because this formation best brings about the slow, circuitous course necessary to warm and humidify the inspired air in such cases. On the other hand, inhabitants of hot moist climates have short noses with nostrils of wide dimensions, and somewhat forward directed, a formation which makes for a straight current and a shorter contact with the nasal mucosa, because there is less need of the warming and humidifying functions.

The dimension of the gateway of the nasal passages can be materially modified by the action of the muscles of the external nose. There seems to be some need of recalling this fact in view of the little importance generally attached to these muscles.

The nasal muscles are of two kinds, those which act to widen the nasal opening and those whose activity is to narrow or close it. The dilator muscles, which are two in number, an anterior and a posterior dilator, run in a general vertical direction over the outside of the vestibule; while the compressor, formed of two parts, takes a general transverse direction. The posterior nasal dilator has one end attached to the maxilla, the other to the posterior part of the alar cartilage; the anterior dilator has only cartilaginous attachments. It extends from the anterior part of the lower border of the lateral cartilage to the edge of the nostril.

It may be that the chief function of these dilator muscles is merely to preserve the necessary tonus to resist the effects of external pressure on the soft parts of the nose and prevent their collapse. In ordinary breathing the negative atmospheric pressure within the vestibule is very slight, but on forced inspiration it is decidedly increased and there is visible depression of the wings if the muscles are inefficient. The alar cartilage which constitutes the boundary of the vestibule is connected by firm fibrous tissue with the lateral cartilage above, and they move inward and outward together under the influences of the muscles. The junction of the two cartilages form an obtuse angle pointing inward toward the septum, constituting the *limen vestibuli* or upper limit of the vestibule. It is a fact of considerable clinical importance that this is the narrowest part of the nasal passage, particularly that it is narrower than the anterior naris.

It was accordingly referred to by Zuckerkandl as the interior naris. The upper insertion of the anterior dilator muscle is just above this line, and when the muscle contracts it acts in a way to straighten out the angle and increase the distance of both cartilages from the septum. The compressor muscle acts in an exactly contrary direction. Anyone who will take the trouble to observe this region when using a nasal speculum cannot fail to note these movements—the sphincter-like action of the compressor muscles often, in fact, proving a great hindrance to satisfactory anterior rhinoscopy.

External nose troubles arise either from diseases or from malformations. Various major and minor dermatologic affections occur not only on the outside of the nose but also within the vestibule because this has a cutaneous lining. The shape of the vestibule favors the retention of secretions within its narrow recesses. In the presence of catarrh, the vibrissæ become matted together, crusts form, and microorganisms, especially staphylococci, are found here in abundance, in contradistinction to the fossa proper, which is normally practically sterile.

Boils, eczema and various obstinate skin affections are, therefore, prone to locate here, and the redness, swelling and deformity they cause, being difficult to conceal, become very embarrassing. They are often also quite painful. Tom Moore has graphically described the sad plight of the sufferer with a certain kind of external nose affection—the pimples nose:

"I never saw a pimples nose  
That told of good things stowed away,  
But brought to mind convulsive throes  
That wracked the wearer night and day."

A furuncle on the external nose, as pointed out by Hurd and others, may not only be painful owing to the sparsity of connective tissue, it may occasionally become a very grave affair. This is due to the fact that venous blood from this region is in part carried by the ethmoid vein, which communicates with the ophthalmic, whence infections may be carried directly to the intracranial sinuses.

Among the more serious affections which have their favorite seat upon the external nose are erysipelas, lupus, papilloma and rhinoscleroma. Erysipelas is due generally to streptococcic infection developing at the site of an abrasion in the vestibule. It may turn out to be a very grave infection. Papillomata of the nasal vestibule, in contradistinction to those which occur deep in the nasal fossa, are small and circumscribed, and do not have the same tendency of recurrence after removal. Lupus begins generally as a small ulcerating tumor



at the anterior end of the septum. It runs a painless course with the formation of scar tissue and eventually may produce deformity of the tip of the nose. Rhinoscleroma, a disease characterized by hard, nodular granulomata in the nasal vestibule, leads to enormous overgrowth and deformity. It is fortunately rare in this country.

Malformations form a large division of external nose troubles. They may be of congenital origin as atresia; arise as results of injury, as the crooked or twisted nose; or appear simply as an exaggeration of type, as the pug nose, the long tip nose, the hump nose or the pinched nose.

The troubles which they cause are of two kinds, (1) disfigurement, (2) dysfunction. It is usually on account of the former that the surgeon is consulted, and often the trouble is more in the imagination of the patient than real.

When malformation is of a degree to constitute a deformity, plastic surgery is indicated. Not only must the surgeon who undertakes this kind of work have a very high degree of skill and originality, he must also have a keen artistic sense. In the correction, harmony of type must be scrupulously preserved. Variations apparently slight can have an unexpected profound effect upon the countenance and personality of the individual. Furthermore, the functions of the external nose muscle must be kept in mind and nothing done which will destroy or disarrange them.

There is one kind of nasal malformation which, it seems to me, has never received from the rhinologist the attention which its importance warrants. I refer to the narrow pinched nose due to alar collapse.

It is often present in young adenoid patients as a result of constant nasal obstruction and mouth breathing, and we find that it generally disappears or greatly improves on removal of the obstructive growths. We see it also in many adults, in some of whom it is difficult to say whether it is a cause or the result of the always accompanying nasal obstruction. We find it with especial frequency in obese persons with weak, flabby muscles, and we get the impression that sometimes it is a part of the general low toned musculature.

It is easily recognized. With each inspiratory act there is a visible sinking in of the nasal wings, whereas normally they should rather expand, or at least maintain their position. With a forced inspiratory act they collapse toward the septum so that a mere chink is left at the nares. The intake of air is labored and accompanied by a sort of whistling or snorting sound or it may be entirely prevented.

To prove that the obstruction is seated, not in the nasal fossa proper, but in the external nose, all you need to do is insert the tip of a nasal speculum just within the nostril and open it so as to fully expand the boundaries of the vestibule. It is often surprising the immediate relief such patients will experience.

Students should always be taught to begin their nasal examination by observation of the external nose and an estimation of the functional capacity of the nares. If this precaution were more rigidly observed it is certain that patients would often be spared operations for turbinectomy and for deviated septum which are unnecessary and of course fail to relieve the complained of obstruction.

An obstruction located at the nares can cause just as much trouble as an obstruction back in the nasal fossa and needs, therefore, just as badly to be corrected. If a patient comes to you complaining that he sleeps restlessly at night, and wakes up in the morning with his throat dry and parched; or if he has bad tonsils, a chronic sore throat, or an obstinate cough, with or without expectoration, do not overlook collapsed nostrils as a possible cause.

The poet Moore, who has already been quoted as an authority of external nose troubles, refers with some feeling to one of the well known effects of the too narrow, though good-looking nose:

"I never knew a maiden face  
With nostrils of the finest mould,  
A nose that might a Grecian grace  
But it would snore when it got old.

I never knew a charming maid,  
With nostrils of the finest bore,  
But when on downy bed she laid  
And slept, she'd always loudly snore."

What shall we do about setting such noses right?

Plastic surgery has not been as successful here as with other kinds of nasal malformation. Attempts to widen the nostril by stripping up portions of the septum and using it as a splint to spread the alæ only have the effect of adding a new obstruction to one already present. Furthermore, any cutting operation on the wing itself is bound to be harmful to the proper functioning of the nasal muscles.

Self-retaining mechanical devices for spreading the nostrils are recommended by some authors. It is possible by a properly adjusted instrument to greatly increase the capacity of nasal respiration. Worn at night it may enable patients to rest comfortably, who would otherwise be much disturbed by laborious asthmatic-like breathing.

The chief objection to the usual instrument of this sort is, in the first place, that it cannot be worn during the day on account of appearances, and secondly, that it causes irritation by pressure against the sensitive septum used as a support.

We have been employing with gratifying result a simple instrument which to a certain extent eliminates these objections. It is bilateral and so constructed that the pressure is away from the septum and against the nasal wings. A single small wire crossing the columella is barely visible.

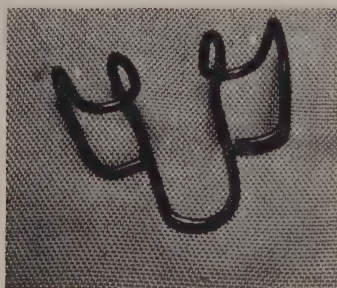


FIG. 1.

Any mechanical device, however, no matter how well made or how completely it overcomes the stenosis, is only a temporary help.

Cannot something be done of a permanent nature, something that goes more to the root of the condition and corrects the underlying cause? We believe that much can be done, at least in those cases in which the pinched nose is due to alar collapse, as a result of partial atrophy of the nasal muscles. The obvious indication is to try to restore the tone and functional efficiency of these muscles.

It is worth remembering that all the muscles of the nose, dilators as well as compressors, are innervated by the seventh nerve, the same that governs the muscles of facial expression and, therefore, presumably like them under a certain degree of voluntary control. This is not at all inconsistent with the theory that the dilator muscles function automatically under the stimulus of the respiratory act, for we see a similar combination of voluntary and involuntary functions in the laryngeal and other muscles connected with respiration.

The nasal muscles may be exercised in two ways: one by willing them to contract simultaneously with inspiration and, secondly, independent of respiration. Both are useful methods and if practiced persistently and systematically will have a decided effect in strengthening these little muscles and restoring their tonus. Practice these exercises

at first in connection with deep respiration; then later without them.

You will find that, independently of the respiratory act, the muscles will contract best against a slight resistance. Place the finger gently against the side of the nostril, alternately back and front, and you can at once or at least with persistence clearly demonstrate separate contraction of the posterior and anterior muscles. Subjects of nasal collapse should practice these exercises for several minutes at a time, several times a day.

If anyone argues that the very slight results to be obtained are hardly worth the efforts, the answer is that very slight results are all that are needed to make the difference between adequate and inadequate nasal respiration, and if this slight difference be bridged it means a tremendous lot to the health and well-being of the patient.

It has not been our object in this brief paper to give a complete account of all the trouble that may afflict the external nose, but rather to call renewed attention of the rhinologist to a part of his specialty that seems to be relatively neglected.

It is sometimes the seat of troubles which are obviously serious; at other times, apparently slight, but important because of the serious consequences which may follow. Troubles of the external nose, therefore, despite their location, are not to be sneezed at.

We have sought to show that the external organ is not only a highly valued feature, one that is regarded as peculiarly expressive of the personality and perhaps also of the intellect of the individual, but that it besides subserves some very useful purposes in the human economy.

An organ thus valued for both appearances and utility is, of course, bound to give the owner some trouble.

But however troublesome, who could afford to be without it? To cut off the nose to spite the face would prove indeed a costly revenge. For the loss of this organ, whether by revenge or from accident or from disease, or as a punitive measure, must be accounted as a calamity of major proportions.



EXTERNAL DEFORMITIES CORRECTED AND REMOVAL  
OF EXISTING INTRANASAL OBSTRUCTION  
ADVANTAGEOUSLY ACCOMPLISHED  
AT SAME OPERATION

By LEE COHEN, M.D.

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It is a well-known fact that many persons desiring correction of external nasal deformities also suffer from more or less marked difficulty in breathing, due to co-existing intranasal obstruction. All rhinologists interested in rhinoplasty agree, therefore, that external deformities should not be corrected while the intranasal obstruction is permitted to remain. Such harmful obstruction should be rectified, not only to furnish comfortable breathing but also because of its ill effects upon general health. Of course, the younger the subject the more pronounced are such ill effects.

For many years it was our custom first to correct intranasal defects by an operation performed six to twelve weeks prior to that done for the external deformity. This method of procedure we found to be a distinct hardship to the patient, not only because he must submit to two operations instead of one, but also because of the increased expense entailed by double hospitalization and added loss of time. In not a few instances objection to two definite operative procedures has been so great as to cause the patient to say, "Oh, just correct the external deformity and leave the inside alone." In a few of our earlier cases, where we were persuaded to do this, it was to our great regret, because of complaints from patients later of inability to breathe properly and their insistence that something be again done for the relief of the discomfort.

For the past three years, therefore, we have been correcting the intranasal condition and the external deformity at the same time, to the entire satisfaction of the patient and ourselves. Besides we find it easier to lower the dorsal plane, when necessary, or correct a lateral deviation of the nose, if a modified submucous resection is done at the same sitting. Only when a rib graft is required in the correction of a saddle nose would we advise against the dual procedure, owing to a greater likelihood of infecting the graft through added traumatization of the tissues. In doing a rib graft it is our fixed rule, after preparing the nose for its reception, to obtain the graft from the rib to shape, and place it in position under the skin of the nose as quickly

as possible, with the least trauma to the nasal tissues. By following this rule we have not experienced a single infection of our grafts. In saddle nose, therefore, any necessary intranasal surgery is done at least six weeks prior to the plastic operation.

Of course, it goes without saying that no plastic surgery should be attempted in the presence of a purulent sinusitis. We must be certain first that such condition has been entirely cured.

In certain types of deformity, such as, for instance, the scoliotic nose, which invariably follows fracture of the nasal bones, we generally find one or both sides of the nose markedly obstructed by septal deflection resulting from fracture and displacement of the septum at the time of the injury. Many of these patients assure us that, whereas prior to the accident they rarely had colds, since that time they have been victims of frequently recurring and tenacious intranasal infections, often accompanied by sinus involvement. In some such conditions of long standing, as a result of these repeated colds, we find also marked hypertrophy of the lower turbinate bodies, with large pendulous posterior ends. These posterior ends not only often completely block the passage of air through the posterior nares, but, by increasing the flow of mucus into the nasopharynx, bring about frequent upper respiratory infections and at times actual disturbance of the gastro-intestinal tract. Besides, by encroaching upon the lumen of the eustachian orifices, interference with the proper ventilation of the middle ear occurs, causing frequent catarrhal exudates as well as purulent otitis media, with or without complications—a well-known danger to the hearing, even to life itself.

In not a few of the long acquiline humped nose cases, we have found a pair of bullous middle turbinates, so large at times as to make constant pressure contact against the septum. Such patients usually complain not only of an almost constant sensation of pressure about the root of the nose and frequently recurring headaches, but many also complain of prolonged attacks of sneezing, accompanied by profuse and annoying hydrorrhea resembling allergic attacks. From such attacks we have seen complete relief follow excision of these bullous middle turbinates in individuals who had been otherwise treated without results.

Although I am fully aware of the almost universally prevailing objection to the removal of middle turbinates, and while heartily agreeing that no needless sacrifice of nasal tissue should ever be made, yet at the risk of having much criticism heaped upon me, I cannot refrain from expressing a conviction, bred of long experience, that many failures to obtain functional results, relief from reflex

headaches and ample breathing space, from intranasal surgery, is due to such conservatism. Where, in dolichocephalic heads with narrow nasal passages, the bullous middle turbinates make pressure contact against the septum, one, or when necessary, both of them, are completely excised by us without hesitation. This procedure has been followed by the most signal benefit to a great number of patients. Many of the cases have been followed up over a period of years, and I am certain that no ill effects have ever resulted from the loss of these middle turbinates.

In many young subjects between sixteen and eighteen years of age, whose difficulty followed injury to the nose in very early childhood, instead of meeting with the usual type of septal deflection, we found that, through fracture with transverse displacement of the cartilaginous septum, accompanied by marked ecchondrosis, both sides of the nasal vestibule were completely blocked. This obstruction appears as an irregular blank transverse wall of cartilage, extending from the outer limit of one vestibule to the outer limit of the other, beyond which it is impossible to see the condition of the interior of the nose. Invariably these young people have a disinclination to eat properly and appear pale and undernourished; they lose sleep because of marked interference with nasal breathing, and show poor concentration in their school or college work. Bimanual palpation over the nasal alæ reveals this type of obstruction as a hard, bulbous mass, and the usual rotundity of the cartilaginous dorsum becomes a flattened surface. It is truly amazing to observe the rapid improvement in these young people after restoration of nasal breathing.

One must not make the error, in correcting this type of obstruction, of removing too much of the obstructing cartilaginous mass, lest there be left insufficient support for the tip of the nose. It is far better to remove submucously from each side of this blank wall enough cartilage to restore sufficient breathing space, and permit the central portion to remain for the support of the nasal tip in the correction of the external deformity.

For the same reason, even in the correction of the ordinarily deflected septum, when external plastic work is contemplated, the classic submucous resection is not a wise procedure. Much difficulty in properly supporting the tip of the nose has been encountered by us in a manner of cases referred by rhinologists for plastic work, after they had previously removed too much of the triangular cartilage in the performance of a submucous resection. By doing the so-called modified resection described by me in the *Annals* of June,

1924, tip support is always assured, without sacrifice of breathing space to an appreciable degree, if at all. This is a very important point and, therefore, even though some of you may be familiar with the procedure, I am here again showing the slide and reviewing briefly the technic:

After having undermined the mucoperichondrium on one side of the septum, as is done in all septal resections, the usual vertical incision through the lower front end of the cartilage is made with the knife, and the mucoperichondrium of the opposite side is undermined. The swivel knife is now passed backward along the upper edge of the vomer until it encounters the bony portion of the septum in the rear, and is then pulled forward in the same line of incision, in order that none of the cartilage be removed. Now with the sharp elevator, the cartilaginous septum is transfixed at its posterior attachment to the bony septum, from which it is completely severed. Thus, you will note, the triangular cartilage is left wholly attached along the bridge of the nose only. At this point, by removing the thick lower portion of the vomer along the intermaxillary ridge, with the bone forceps, space is made to allow the curved or deflected triangular cartilage to straighten out. With this accomplished one may proceed with the operation, removing as much of the deflected bony portion of the septum as may be necessary to obtain a smooth, straight septum in the rear.

In an extreme deflection, where obtaining a straight septum seems almost impossible without removing practically the whole of the bony septum, another word of caution is offered, lest through removal of too much bone we later have a flail membranous septum without bony support, which flops to one side on inspiration—impairing the ingress of air through that side—and flops to the other side on expiration, impairing the egress through the opposite side. I know of nothing in the nose more difficult to correct than this type of flail membranous septum, or nothing which causes the individual more annoyance through its marked interference with nasal breathing.

To prevent such an occurrence, only the thickest portion of the bony septum should be removed. Then, with the Struyken scissors, the thin upper portion, which is made up of the perpendicular plate of the ethmoid, is severed from its attachment to the base of the skull, so that it may be dropped down into the pocket between the septal membranes. This transplanting of the perpendicular plate to the lower portion of the septal pocket absolutely precludes the occurrence of the flail septum, and at the same time, through thinning of



the upper half of the partition, makes for greater space in the upper narrowest portion of each side of the nose. Of course, the intermaxillary ridge may be removed with the hammer and chisel, as is usually done, but as much of the vomer bone as possible should always be retained, even if severed from its attachments, to aid in supporting the septal membranes.

So far as hypertrophied lower turbinated bodies are concerned, we endeavor to excise as little of the soft tissue as possible, and rarely if ever any of the bone itself. We prefer infracturing the bones and



FIG. 1. Schematic drawing indicating incision in triangular cartilage, used in *modified submucous resection*.

forcibly pressing them outward against the antral walls, removing only the enlarged pendulous posterior ends with the snare. This furnishes sufficient breathing space in the vast majority of cases, but should it occasionally not do so, it is an easy matter a few weeks later, as an office procedure, to reduce the still swollen turbinate body, either by galvanocauterization or by the use of the high-frequency current.

We have performed the dual operation in thirty or more cases, with uniformly satisfactory functional and cosmetic results. Time in the hospital averages five days—the same as when the external deformity alone is corrected—while postoperative care in the office is not prolonged beyond that accorded a simple rhinoplastic case.

Our technic can best be made clear by describing in detail a few of our typical cases:

## REPORT OF CASES

*Case 1.*—F. R., age twenty-nine, first consulted us September 22, 1934. About seven years prior she was in an automobile accident, sustaining an injury to the nose. Examination showed a convex vertical deformity, fairly well marked hump on the dorsum, and a rather over-sized cartilaginous nose with drooping tip. Directly to the left of center of tip was a scar about one-half inch long, caused by a cut from the broken windshield. Internally we found a rather marked obstruction, due to deflection of septum to the right, along with prominent hypertrophied and intumescent lower turbinates with large pendulous posterior ends. Patient complained of great difficulty in breathing and was highly conscious of the deformity.



CASE 1.

On November 19, 1934, under local anesthesia, augmented by three capsules of three grains each sodium amytal, a v-shaped section was removed from the subseptum. The base of this section, about 10 mm. in width, lay at the dorsum nasi just above the tip and the apex at the anterior nasal spine; through the defect thus created, the membrane on each side of the septum was undermined, and a modified submucous resection performed. Double lower turbinate compression followed, and both enlarged posterior lower turbinate ends were removed with snare. This having been accomplished, both nasal cavities were lightly packed with sterile nu-gauze tape, before proceeding with the correction of the external deformity.

A bilateral incision within each vestibule at the junction of each ala with the bony nose was now made, and joined over the top of the septum nasi. These incisions included the lining of the vestibule and overlying cartilage up to the skin covering the bony nose, which was undermined in its entirety. The irregularities and hump on the bony dorsum were now removed with rasp. Closing the subseptal defect with four black silk sutures on each side elevated the tip and shortened the nose.

To shorten the sides of the nose, v-shaped sections were now cut from the upper edge of each ala incision, to the extent that they overlapped the lower edges, but left attached at their bases where they join with the mucous membrane of the septum. The apices of these attached triangular flaps, together with one-third of their length, were cut away and the remaining two-thirds turned upward over the septum of the nose, with their raw surfaces apposing, and fastened in this position with one black silk mattress suture. These flaps so placed served to obliterate an existing depression of the dorsum just above the tip, which otherwise would have marred our result.

After removing the sterile tape temporarily placed, the interior of the nose was again packed loosely with one-half inch iodoform nu-gauze tape, between the septum and middle turbinates. Two soft cigarette packs on each side to



CASE 2.

support the septum (as we do in regular submucous operations) were now placed in the lower portion of each nasal cavity and the vestibule filled with iodoform tape, the lower ends of which were carried over the columella from one vestibule to the other three or four times, to protect the subseptal wound. The copper saddle splint was now applied in the usual way.

Forty-eight hours later all packs were removed, but the vestibule was again loosely packed as before with iodoform tape. On the fifth day all packs were left out of the nose, and on the seventh the splint was removed, the skin bathed with 95 percent alcohol, and after removing all intranasal sutures the splint was again applied. From now on the splint was changed every fourth day (in order to take care of the skin) for three weeks, when it was discontinued.

*Case 2.*—Milton S., age twenty years, was seen by us October 31, 1933, giving the following history: At the age of eight, while riding on the rear seat of a motorcycle, he fell and fractured his nose. After swelling subsided, the nose was seen to be very crooked and displaced toward the right side. For the past five or six years he experienced great difficulty in breathing through the





CASE 3.

nose, was annoyed by frequent attacks of sneezing and more or less continuous postnasal dripping. He also suffered often from severe headaches.

The external nose was of the scoliotic type, being displaced also toward the right cheek, and the bony dorsum was made irregular by exostoses along the line of fracture. Internally the nose was greatly obstructed by an S-shaped septal deflection, the cartilaginous portion curving greatly toward the left, while the bony portion bent toward the right side. On the summit of the bony deflection there was a well-marked ledge where the vomer and perpendicular plate of the ethmoid joined. Markedly hypertrophied lower turbinates, along with a



CASE 4. Rib graft.



large bullous left middle turbinate, served to make almost a complete obstruction.

On November 1, 1933, at Sinai Hospital, patient was operated upon under local anesthesia, augmented by sodium amytal and twilight sleep.

A modified submucous resection was first performed through the usual incision, the septum straightened and the thickened ledge removed. Compression of both lower turbinates followed, forcing them outward against the antral walls, and removal of the posterior pendulous ends with the snare. Now the large bullous left middle turbinate was completely excised and the nasal cavity packed loosely with nu-gauze tape, preparatory to correcting the external deformity.

The usual bilateral intranasal incisions were now made along the junction of each ala with the bony nose, and through these the skin over the entire bony dorsum was undermined. All irregularities of the dorsum were smoothed with the rasp, before completely mobilizing the nose. by severing with the saw the nasal bones at their attachment to the nasal processes and infracturing their attachment to the frontal bone with hammer and buffer, and Carter's bone forceps.

The nose was thus rendered so flail that it could be placed in any desired position toward the midline of the face, without any tendency to spontaneous displacement. Temporary packing was now removed, and the usual iodoform tape and cigarette packing inserted, after which the splint was applied.

This patient returned to his home in Pittsburgh after four weeks with a very happy cosmetic and functional result.

In closing. I would emphasize that from our experience where plastic work is required, the welfare of the patient is best served when the plastic operation for external deformities and the necessary correction of intranasal obstructive conditions are considered as a joint procedure.

## A CONSERVATIVE OPERATION FOR CHRONIC MASTOIDITIS

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The faulty practice of defining as *otitis media purulenta chronica* cases of chronic otorrhea which have persisted in spite of treatment and in which operation is indicated is quite apparent. The condition should be termed a chronic mastoiditis, and deserves to be considered as such and not as the name implies merely a chronic purulent discharge from the middle ear. The site of the trouble and source of the discharge lies not in the middle ear but in the mastoid process itself. As we well know there usually forms in chronic mastoiditis a cavity of varying size in the region of the antrum, which becomes filled with granulations, cholesteotoma, pus, detritus, dead bone, etc. During the course of the disease the overlying mastoid bone and surrounding walls become rarefied, hard and condensed, the accumulated products of inflammation forming in this antral cavity a nidus of infection, which constantly reinfects the middle ear, causing a continuance of the discharge.

In chronic mastoiditis pus in this antral cavity may form under pressure and be so gradually applied as to give little evidence of an erosion of its bony walls which may suddenly take place at any time and the infection attack the contiguous dura of the middle fossa above, knee of the sinus behind and externally, or the lumen of the external semicircular canal below.

When intracranial complications develop in the course of chronic mastoiditis affecting the brain, sinus or labyrinth, it is in these specified localities that the initial evidences of infection are most often found, caused no doubt by the proximate relation of the knee of the sinus, cap of the external semicircular canal and the under surface of the dura of the middle fossa to this cesspool of infection usually found in the antral region in chronic mastoiditis.

Owing to the mal-development commonly found in the mastoid process of patients afflicted with chronic mastoiditis the relation of these important structures, the antrum, the knee of the sigmoid sinus, the dura of the middle fossa and the cap of the external semicircular canal, is likely to be much closer, more intimate and atypical than is the case in a normally developed bone. It is evident, therefore, that

the only way surgically to insure a patient suffering from chronic mastoiditis from serious intracranial complications is to thoroughly aerate, drain and permanently expose the antral region as otherwise this infected focus is apt to remain or reassert itself. Nature sometimes has a way of spontaneously curing chronic mastoiditis by causing a necrosis of the inner extremity of the superior bony canal wall, this of course forming the external attic wall.

The necrosis often includes the epitympanic ring. When a sufficient amount of bone has sloughed away in this region direct drainage is then permitted from the interior of the antrum cavity to the external auditory canal. This improved direct drainage frequently effects a spontaneous cure in chronic mastoiditis. The results in these cases of spontaneous cures often approximate those obtained by the operation about to be described. It might be said that this conservative operation which we have practiced is but an extending of the effort that Nature at times makes to cure chronic mastoiditis.

We find the middle ear will return to normal in so far as is possible when once this antral region has been permanently aerated, drained and healed. Good sized perforations of the drum, both central and marginal, will frequently undergo repair. There seems to be a primitive effort on the part of the tympanic membrane to reconstruct itself when once the cause of this destruction, *i.e.*, the antral abscess, has been healed.

In recent years I have limited the application of the radical operation to those cases in which intracranial or labyrinthine complications are threatened or present and when loss of hearing or destruction or malformation of bone is so great in the region of the antrum and middle ear that no useful result could be obtained by performing the conservative operation. In 1912 I performed my first conservative operation, at which time I believed that it would, when indicated, do as much for the patient suffering with chronic mastoiditis as the radical operation and in addition not alone save, but in many instances, improve, the hearing. Today after twenty-three years experience with the conservative operation I wish to reaffirm this opinion. In 1912 I was unaware of an article by Bondy published in 1910, describing an operation more nearly approaching the one about to be depicted than any other designed to accomplish the same result. Since the radical mastoid operation was first performed its dominating thought has always been to secure a dry postoperative tympanic cavity. Any useful hearing that the patient might retain in the operated ear was an unlooked for blessing and usually of little practical importance as compared with the unoperated ear. In this con-

nection it is interesting to note that one rarely sees a patient upon whom a double radical mastoid operation has been performed. This can hardly be due to the scarcity of patients upon whom the double operation is indicated and might be performed but rather in my opinion to the reluctance on the part of the otologist to subject the individual case to the risk of the great loss of hearing which such an operation might entail. I have no such hesitancy in recommending the performance of a double conservative operation. I have performed the double conservative operation in many instances and all of the patients were subsequently able to continue various occupations as salesmen, stenographers, office workers, etc., and have claimed that their hearing was materially improved.

Unlike the usual radical mastoid the conservative operation which we recommend is not primarily intended as one which would produce a dry tympanic cavity but to obtain first a useful ear and then if possible a dry one. It will be noted in the description of this conservative operation that the middle ear structures, membrana tympani, ossicles and lining mucous membrane are allowed to remain practically undisturbed. This being true it follows that in some cases where there is a large perforation in the drum which remains unhealed there may subsequently be some mucoid discharge; this exudate is the natural secretion from the mucous membrane which is allowed to remain lining the tympanic cavity. It will be seen that the tympanic cavity does not heal by epidermization as is the case after a radical mastoid operation is performed, but by the restoration of normal mucous membrane, while throughout the rest of the cavity epidermization takes place as in the radical operation. Any post-operative discharge which may occur after the wound has healed is usually intermittent, non odorous, mucoid in type and unnoticed by the patient. As long as the antral cavity is permanently aerated, healed and dry, this discharge, if present, has no surgical significance.

*Operative Indications.*—I. This operation is indicated in any case of chronic mastoiditis with good hearing in which the ear continues to discharge despite conscientious and persistent treatment, as it is reasonable to suppose that a continuation of the discharge will cause a diminution of hearing in the affected ear.

II. It is also indicated in cases similar to the above where the hearing is not so good but with the drum more or less intact.

III. It is indicated in cases of recurrent mastoiditis where the posterior auditory canal has collapsed producing the effect of a slit-like opening.



IV. It should be the operation of choice in cases of exostosis of the canal associated with chronic mastoiditis and in perforation of the premastoid lamina with necrosis of the posterior bony canal wall.

V. This operation is especially indicated in children and young adults with chronic mastoiditis in whom treatment has proved unavailing.

*Anatomical Considerations.*—At this point it may be well to review the surgical anatomy of the epitympanic region. The attic of the tympanum is that portion of the middle ear cavity which lies above the level of the tympanic membrane. It contains the head of the malleus and the greater part of the incus. These two ossicles articulate in about the middle of the attic. Running upward from the head of the malleus to the vault of the attic is the superior ligament of the malleus. The malleo-incudal body, with the superior and anterior ligaments of the malleus, divides the epitympanum, surgically, into two cavities—the internal attic lying internally to these structures and the external attic, lying externally. The external attic is still further subdivided by the external lateral ligament of the malleus into Prussak's space which lies below, and a much larger space, above, the external attic proper. It will be seen that in chronic mastoiditis the avenue of escape for pus and inflammatory products is indirectly by way of the external and internal attic to the tympanic cavity proper, thence through the drum to the external auditory canal. It will be noted that the inner extremity of the superior bony canal wall to whose inferior margin is attached Schrapnell's membrane and the membrana tympani, also constitutes the external attic wall and acts as a distinct barrier to the direct escape of inflammatory products from the mastoid, antrum and attic. Until a necrosis occurs in this superior wall with a subsequent dehiscence producing in effect an attic perforation or its artificial removal by operation takes place, pus from the mastoid cannot drain directly but must pass through the middle ear before reaching the auditory canal.

The essential distinguishing feature of this conservative operation is the complete removal of the wedge-shaped mass of bone constituting the superior bony external attic wall, and superior bony canal wall permitting direct drainage from the interior of the mastoid. It is removed in its entirety, posteriorly from the fossa incudus to a point in front where it joins the anterior bony canal wall. Roughly speaking this could be represented diagrammatically by using the face of a clock as an illustration and removing that portion of the dial represented by the space lying between the figures eleven and three.

*Operation.*—This operation was described in a paper which I read before the American Otological Society in 1916 and subsequently depicted in the Transactions of the 34th Annual Meeting of the American Laryngological, Rhinological and Otological Society in 1928. The usual post-aural incision is made with the exception that its upper extremity is curved until directed horizontally forward, terminating at a point about an eighth of an inch above and in front of the superior attachment of the auricle. The soft parts anterior to this incision are elevated and retracted forward. The mastoid cortex is removed with a gouge and the subcortical cells if any, with curettes until the antral space is opened.

The bone in the zygomatic region is removed, the posterior bony canal wall lowered and the antrum curetted and widened to its fullest possible extent until the cap of the external semicircular canal becomes plainly visible. Next the external attic wall is removed by passing a small, narrow Spratt curette from the antrum behind directly forward until the cutting edge of the spoon rests immediately on the internal aspect of the bony external attic wall.

The long axis of the curette during this maneuver should be passed parallel to the same plane as the external semicircular canal and held slightly above it. By observing these precautions we insure the entrance of the instrument into the external and not the internal attic, which mistake would result in the unintentional removal of the incus. With the spoon in the external attic in the position as described the short process of the incus lies directly below and external to it and by keeping the edge of the curette in close apposition to the internal aspect of the bony external attic wall there is no danger of disturbing the malleo-incudal body lying in front and internal to the back of the end of the curette.

The external wall of the attic is then removed by curetting directly outward, the direction of motion being still in the same plane as the external semicircular canal. The posterior superior bony canal wall is lowered to within about one-eighth of an inch of the membrana tympani and reduced in thickness until the facial ridge is reached, leaving only the epitympanic ring arching above the top of the drum from the posterior to the anterior canal wall. The short process of the incus should now be visible; it is commonly found imbedded in a mass of granulation tissue but is always located just below and anterior to the cap of the external semicircular canal resting in a slight hollow termed the fossa incudus, to which it is attached by a ligamentous band, the posterior ligament. Care should be taken not

to rupture it as the incus would probably become dislocated subsequently.

We now continue to remove the external attic wall by curetting in the aforesaid manner until the body of the incus and the head of the malleus is exposed. During the latter stage of this portion of the operation the curette has been in contact with the anterior wall of the attic. The epitympanic ring is now removed and granulations, polypi and cholesteotoma lying in the internal and external attic are removed by curetting on either side of the malleo-incudal body, care being observed not to destroy the suspensory ligament of the malleus. The drum and ossicles are of course not removed but left in position and disturbed as little as possible during the course of the operation. The wound in the middle ear is now gently syringed. On conclusion of the bone work an L-shaped meatal flap is cut as in a radical operation, the cartilage removed and the flap sutured above to the temporal fascia. Light iodoform gauze packing is lightly placed in the antral region and the posterior wound is sutured throughout. No packing is placed in the tympanic region either now or subsequently as in a radical mastoid operation for fear of dislocating the middle ear structures which have been left lying *in situ* in the tympanic cavity.

*Postoperative Care and Dressing.*—As differentiated from those upon whom a radical mastoid operation has been performed it is necessary to do a complete dressing every day beginning the day following operation on those patients who have undergone this conservative operation as otherwise some infection of the soft parts is apt to develop. Only the antral region should be packed and when cleansing the wound we must remember that the remains of the drum and ossicles lie at the bottom of the cavity and can be easily dislocated, so that care should be taken not to do so.

The bandage is worn until the posterior wound heals, when it is removed. The packing is then usually continued daily, only however, in the antral region in such a manner as to leave some access of air to the middle ear, this is continued usually until the antral region epidermatizes, when dry treatment is instituted. The antral space should not be permitted to fill with granulations and should be kept free until epidermization takes place.

#### SUMMARY

I feel that this operation is conservative as compared with the usual radical mastoid operation because of its beneficial effects upon the patient's hearing. Facial palsy which sometimes follows the

usual radical mastoid operation is more frequently produced by the necessary intratympanic instrumentation than is commonly supposed. You will note that the danger of producing a similar complication in the same manner while performing a conservative operation is practically nil as the middle ear structures are undisturbed. The complete removal of the bridge of bone forming the external auditory canal and constituting at the same time the inner extremity of the superior bony canal and the external attic wall, to which Shrapnell's membrane and a part of the drum on either side is attached, is a most important factor in securing subsequent direct drainage from the antral region and epitympanum. Its removal apparently does not weaken materially the support for the drum.

We consider that one of the greatest fields for this operation is in children and young adults afflicted with chronic mastoiditis.



## SUBCUTANEOUS EMPHYSEMA AS A COMPLICATION OF FOREIGN BODY IN A BRONCHUS

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The spontaneous occurrence of subcutaneous emphysema in a case of foreign body in a bronchus prior to bronchoscopy or other surgical procedure is an alarming complication. Subcutaneous emphysema of the neck, extending at times to the face and trunk, is not uncommon after tracheotomy. It is observed in wounds, injuries or surgical operations, of the esophagus, the upper air passages, larynx, tracheobronchial tree or lungs. The development of subcutaneous emphysema as a complication of pertussis, diphtheria and pneumonia in children has been reported; it also was observed in cases of influenza during the epidemic of 1918.

In 1922, Wiskovsky and Camrda<sup>1</sup> reported the occurrence of emphysema of the mediastinal and subcutaneous tissues following aspiration of a bean into the right bronchus. Forbes<sup>2</sup> recorded the development of extensive subcutaneous emphysema in a child, three days after a peanut had been aspirated. In Woodward's<sup>3</sup> case, a child aged six years, subcutaneous emphysema occurred following the aspiration of a grain of corn. In one of three cases reported by Vinson and Moersch,<sup>4</sup> the foreign body, a paper clip, was believed to have penetrated the bronchial wall. In the remaining two the foreign bodies were peanuts. In both, there was emphysema of the subcutaneous tissues of the neck, head and upper thorax; in one there also was air in the pericardial sac. In the case of a child, aged three years, reported by Carpenter,<sup>5</sup> emphysema occurred three days after choking on a peanut. The involvement was very extensive, including the scalp, face, neck and entire trunk down to the pubes.

In all of these cases the emphysema occurred prior to attempts at removal. In none was there an associated pneumothorax. In all the emphysema cleared promptly following bronchoscopic removal of the foreign body and recovery was complete without mediastinal or pulmonary complications.

A fatal case of foreign body in a bronchus accompanied by spontaneous emphysema and complicated by a suppurative mediastinitis and pericarditis was reported by Imperatori.<sup>6</sup> Two days after choking on a peanut the child developed swelling of the tissues of the

neck. This extended to the subcutaneous tissues of the chest and seemed to increase after each paroxysm of coughing. The findings by physical examination and roentgen study showed changes in the lower lobe of the right lung with subcutaneous emphysema of the head, neck and chest; also involvement of the mediastinal tissue. At bronchoscopy a peanut was removed from the middle lobe bronchus and an additional small fragment from the lower lobe bronchus. Postoperative improvement was temporary; the patient died about ten days later from suppuration involving the mediastinum and pericardium. At autopsy nothing was found to explain the emphysema. It was suggested that there was a break in the party wall between the trachea and the esophagus.



FIG. 1. Photograph of child aged twenty-one months, with extensive emphysema of tissues of head, neck, upper chest and mediastinum. The size of the head and neck was enormous in contrast to the remainder of the body. The eyelids were swollen completely shut. The quantity of air in the tissues was so great that characteristic crepitation could be elicited only along the junction of the normal and emphysematous tissues.

#### REPORT OF CASES

Two cases of bronchial obstruction produced by foreign bodies complicated by subcutaneous and mediastinal emphysema have been observed by me. These are of interest because of the extent of the emphysema, the rapidity of its occurrence, the diagnostic problem involved in one, and the prompt clearing up of the emphysema following removal of the foreign body.

*Case 1.*—A child, aged twenty-one months, developed swelling of the left cheek secondary to a tooth infection. Five days later there was cough with fever (104° F.). The family physician suspected measles. Fever persisted.

On the twelfth day of the illness following a severe paroxysm of coughing there was noted swelling of the neck. This spread rapidly to the face and chest, being more marked on the right side. Dr. D. F. Daley was consulted and found evidences of partial obstruction to the left bronchus with subcutaneous emphysema of the face, neck and chest. On questioning the family he learned that the child had eaten a whole cooked prune at the onset of the illness and more recently had been given peanut candy. There was, however, no history of choking or gagging.

On admission to the Bronchoscopic Clinic, Jefferson Hospital, about twenty-four hours after the onset of the emphysema the child seemed comfortable when undisturbed. There was no dyspnea nor wheeze. The head, neck, shoulders and upper chest were enormously swollen. The eyes were completely closed (Fig. 1). The skin over the swollen areas appeared glossy, pallid and tense. Attempts to palpate or percuss the chest over the emphysematous area provoked



FIG. 2. Roentgenograms made at the end of inspiration and expiration show moderate obstructive emphysema of the left lung. The mediastinal emphysema is quite extensive. There is roentgen evidence of air in the tissues along the left border of the heart. The subcutaneous emphysema of the neck, about the shoulders and the lateral aspects of the chest is clearly shown. (Films by Dr. W. F. Manges.)

crying, suggesting that this was painful. Dr. T. McCrae saw the child in consultation but could not make a satisfactory examination of the chest on account of the emphysema, which extended to the fourth rib anteriorly, in both axillae laterally, over the upper back, and also because of the distress produced by movement of the child. The signs over the lower back, the only area that could be properly examined because of an absence of emphysema, indicated that there was partial obstruction to the lower lobe of the left lung. The roentgen findings reported by Dr. W. F. Manges corroborated this. In addition to the obstructive emphysema of the lower lobe there was observed emphysema involving the mediastinal tissues (Fig. 2).

It was noted that there was a progressive increase in the extent and degree of the subcutaneous emphysema following the admission of the patient to the clinic. Although bronchoscopy was believed to be hazardous, the findings by physical examination and roentgen study and the increasing subcutaneous em-



physema were considered to be of sufficient importance to warrant its being done without delay. At bronchoscopy a portion of peanut was removed from the left main bronchus. It was observed at this time that there was marked forward bulging of the posterior pharyngeal wall, indicating a retropharyngeal accumulation of air; the trachea also was narrowed. The larynx appeared normal. Recovery was uneventful. The subcutaneous emphysema cleared promptly and the patient was discharged well ten days following bronchoscopy. A roentgen study at that time indicated that the mediastinal emphysema had cleared and that both lungs functioned normally.

*Case 2.*—A child, aged fourteen months, choked while eating peanuts. About twenty-four hours later, following a severe paroxysm of coughing attended with cyanosis there was noted swelling of the right side of the neck and face. On admission to the Bronchoscopic Clinic, Jefferson Hospital, two days following the accident, there was observed marked subcutaneous emphysema of the neck, head and upper chest, being more marked on the right side and over the upper end of the sternum (Fig. 3). The physical signs indicated that there was obstruction to the bronchus of the right lower lobe. This was corroborated by roentgen study; however, there was no obstructive emphysema present. There was roentgen evidence of emphysema of the mediastinal tissues. Following removal of the foreign body, a peanut, the emphysema promptly cleared.

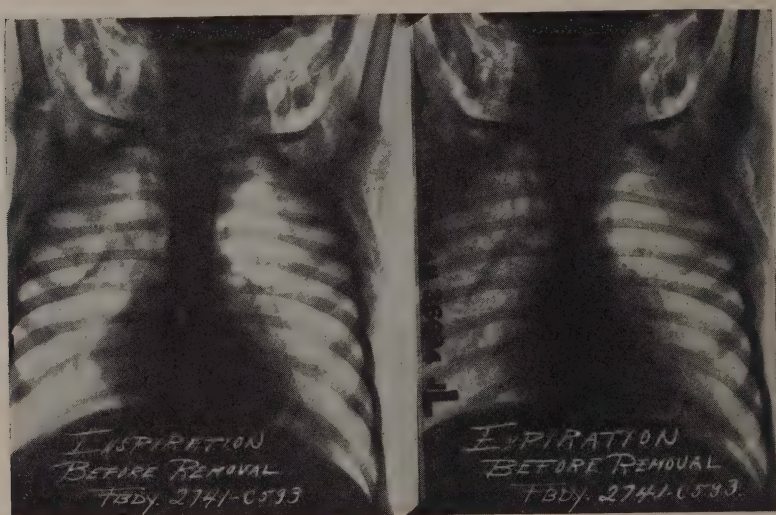


FIG. 3. Photograph of child, aged fourteen months. Prominence of the air-containing tissues about the front of the neck and over the upper end of the sternum is clearly shown. The tissues of the right side of the face were more involved than those of the left. The right eye could not be opened.

#### COMMENT

There was no roentgen evidence of pneumothorax in either case, although air could be demonstrated in the mediastinal tissues in both. The extent of the emphysema rendered physical examination difficult in case No. 1; the history was misleading, but the physical and roentgen findings were sufficiently positive to warrant a diagnosis of partial



obstruction of the bronchus. The emphysema did not lessen in degree or extent until after the foreign body was removed. In case No. 1 there was an appreciable increase of emphysema up to the time of bronchoscopy.

The exact mechanism of production of emphysema in these cases is not known as none have come to autopsy. It is interesting to note that of the seven cases previously reported and the two cited herein eight of the foreign bodies were of vegetal origin. These were incapable of producing direct trauma to the wall of the bronchus as might occur with a penetrating foreign body. In the case of paper clip in the bronchus with emphysema,<sup>4</sup> the pointed end was believed to have penetrated the bronchial wall. If this is the factor necessary for the production of subcutaneous emphysema it should be a common complication in cases of safety pins or other pointed metallic foreign bodies in the bronchi. On the contrary, it is rare.

An interesting study concerning the occurrence of subcutaneous emphysema in a group of fatal cases of pneumonia was reported by Clark and Synnott.<sup>7</sup> A total of twenty cases was observed. Of these, three were carefully studied at autopsy. The common distribution of the "gas" was about the base of the neck anteriorly and laterally and over the upper portion of the chest to about the third rib. In two cases it was more extensive, being distributed in the tissues of the cheeks and eyelids and in small areas over the sides of the chest and abdomen. At autopsy this distribution was corroborated. In the marked cases there was evidence of "gas" in the vascular sheaths of the arteries of the upper extremity extending as far as the thumb. It was also found in the mediastinal tissues, beneath the pleura at the hilum of the lung and in the smaller pulmonary septa. It was not observed in muscle sheaths. Small bits of fascia containing "gas" were found negative for aërobic and anaërobic bacteria. They reported that the air-containing alveoli appeared emphysematous in the more nearly consolidated lung lobes. The distribution of the "gas" in the tissues corresponded with the dissemination one would expect as it escaped under pressure from the weakened alveoli of the lung.

The similarity in the distribution of the air in these and in the foreign body cases is striking. It is probable that the underlying mechanism is identical. Obstructive emphysema is a common clinical finding in vegetal foreign body cases. In the presence of a severe paroxysm of coughing distended air vesicles in a case of marked obstructive emphysema may rupture, forcing air into the lung tissues. The leakage of air lessens the degree of obstructive emphysema, but the valvular obstructive mechanism in the bronchus remains so that

a certain quantity of air is forced into the tissues through the ruptured alveoli during each expiration and particularly when the patient cries or coughs. Air forced along the hilum of the lung into the mediastinum finds its way through the upper aperture of the thoracic cage to the tissues of the neck, face and trunk. The extent and degree of the subcutaneous emphysema are dependent in great part if not entirely on the effectiveness of the valvular action of the foreign body. With removal of the foreign body, air contained in the bronchi can no longer be forced through the ruptured alveoli and the emphysema subsides. The air in the tissues is promptly absorbed.

In conclusion, it can be stated that the fundamental requirements for the production of subcutaneous emphysema complicating bronchial foreign body are rupture of overdistended alveoli such as occur in obstructive emphysema through an increase in the intra-alveolar tension as is observed in paroxysms of coughing. Air entering a bronchus must be trapped during expiration. The degree and extent of the dissemination of the air in the tissues are dependent on the effectiveness of the valvular action of the foreign body. Prompt removal of the foreign body is indicated to prevent further extension of the emphysema and the possible development of complications. The occurrence of mediastinal suppuration results from forcing infected secretions contained in the obstructed bronchus through the ruptured alveoli.

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## ESOPHAGEAL DISTURBANCES

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More than two thousand years ago the Father of Medicine noted that nervous women often complained of a lump in the throat. Galen's translation of Hippocrates has made the term *globus hystericus* familiar to us all. Perhaps each of us has noticed in moments of great emotional stress a sensation of tightness in the throat. However, *globus hystericus* has been somewhat over-emphasized, granting the frequency that it is met with, in this respect what was a good enough diagnosis in the Hippocratic day is not an adequate one in 1935. In the great majority of the cases of esophageal cancer, for example, the patient has been treated many months for globus with sedatives and belladonna until it became too late to do anything.

Let us simplify this discussion by dividing esophageal obstructions into those due to extrinsic and intrinsic factors. At the first extrinsic cause may be listed retropharyngeal abscess. This condition is of acute onset and is readily diagnosed. Last month I saw in consultation such a case in which the rapid progression of the abscess had shut up the windpipe, necessitating an emergency tracheotomy with intravenous and rectal feeding for two days. The treatment of retropharyngeal abscess, of course, is prompt and efficient drainage. If it is secondary to perforation of the esophagus by a foreign body, obviously the removal of the foreign body is essential. Mediastinitis, which may also be secondary to a foreign body from the esophagus or tracheobronchial tree, is even more serious.

Obstruction may arise from direct infiltration into the esophagus or the tissue surrounding it by carcinoma of the larynx, thyroid or other neighboring structures. Retrotracheal or substernal adenoma of the thyroid and tumors in the mediastinum primary or metastatic, are also possible causes.

Aneurysm of the arch of the aorta not infrequently causes partial obstruction. Blackford in 1932 reported a case of right aortic arch which interfered with the course of the esophagus and called attention to other aortic anomalies as possible causes of obstruction. Although these factors do not intimately concern the bronchoscopist it is important always to bear in mind that they may be responsible and it is, therefore, essential that the patient with symptoms of esophageal obstruction should be carefully studied by an internist and a roentgenologist before resorting to esophagoscopy.

By far the most common intrinsic cause of obstruction is benign cicatrical stenosis. Cicatrical stenosis in the majority of cases results from swallowing of lye. Too often small children in the poorer classes, particularly among negroes, will drink a solution of lye by accident. Other caustics will have the same effect and they may be taken by an adult with suicidal intent. The resulting stricture is apt to be greatest at one of the three anatomic narrowings of the esophagus, particularly the cricopharyngeal, and there is often more than one. Of course, the best treatment is preventive, namely, to keep caustics out of the reach of children, to keep all poisons in distinctive containers and to keep the adult population in a healthy frame of mind. The history in perhaps 90 percent of cases will make clear the diagnosis. In my opinion, the best method of treatment is to perform immediately gastrostomy and, after the acute chemical esophagitis has subsided, to begin retrograde dilatations by the method of Tucker. It is important to remember that the dilations must be kept up at intervals over a period of years.

In about 10 percent of cases, cicatrical stenosis is a sequel of typhoid fever, diphtheria, or tuberculosis. The vomiting of pregnancy has not infrequently been blamed, and stenosis has occasionally been attributed to syphilis. In this group, with a slow onset, one can often have the patient swallow a twisted silk string and dilate the esophagus from above, but Trousseau said many years ago, "Sooner or later all cases of stricture of the esophagus die of the bougie."

An uncommon cause of obstruction is the lodgment of a foreign body. I recently reported a case in which a textile bobbin had been arrested just above the left bronchus, eventually causing complete occlusion. This child had been kept alive many months by means of a gastrostomy tube. Finally I succeeded in dislodging the bobbin from its dense scar tissue with sharp dissection and extruded it into the stomach. The ignorance and poverty of this child's parents interfered with a complete course of dilatation, but more than two years after the removal of the bobbin her deglutition seemed essentially normal. The moral of this case is that one should always take seriously a child's story of swallowing a foreign body, no matter how preposterous it may sound.

Peptic ulcer of the esophagus and esophagitis have been stressed by Jackson, and the association of peptic ulcer of the esophagus with ulcer of the stomach or duodenum, has been stressed by his son. The diagnosis should be suggested by pain on swallowing. The treatment is mainly dietary, though topical applications may be required, and after healing, dilation is often necessary.



Let me warn you against the great danger of passing a bougie blindly, or even attempting to pass a bougie at all from above, even on a guide string, if the stricture is marked.

Cancer of the esophagus may be easily diagnosed early, it is of slow growth and late metastasis and yet is invariably fatal. The greatest factor in this mortality is late diagnosis. Chevalier Jackson explains this on a basis of ignorance in the medical profession. We have been taught that the most important factor is the history, but it has been proven that this is not so. Many patients give a history of dysphagia over a number of years. If a precancerous condition had been recognized and properly treated, practically all of these might have been saved. Another point that Jackson has emphasized is that malignant disease of the esophagus is always intermittent in its obstructive symptoms. It is recognized that after the general examination and the roentgenologic examination of the barium-filled esophagus, the next step is to look at the lumen of the organ. It is more than possible that early examination would permit the removal of the first clump of malignant cells when no more than a few millimeters in diameter. In all cases, of course, biopsy is necessary. One of the most important things to exclude is hiatal hernia of the stomach.

It must be emphasized again that the history of *globus hystericus* cannot be distinguished from the early history of esophageal cancer, and that an esophagoscopy constitutes no risk whatever to the patient. Carcinoma of the esophagus occurs with increasing frequency as the cardia is approached.

Squamous cell carcinoma is the most common form of cancer of the esophagus, but adenocarcinoma ranks a close second. Sarcoma is comparatively rare as are also benign tumors.

Esophageal cancer grows slowly. Jackson has stated positively that "a cancer in the esophagus that has reached the stage of complete obstruction of the lumen has been present for at least a year." In the absence of metastases it is theoretically possible to remove the entire organ, but this, insofar as I know, has not been done successfully. With the rapid advances being made in the surgery of the thorax, we may feel sure that esophagectomy will be accomplished, but at the present time it would appear that our best hope is palliative treatment. One great aid in palliation is the absence of pain in cancer of the esophagus. This not only spares the patient suffering and loss of sleep, but it also makes opiates unnecessary. The cooperation of an internist in seeing that the patient is fed regularly a diet adequate in both calories and vitamins is most important. It is, of course, of the utmost importance that the food should be in a finely

divided state and also that the patient must still attempt to chew in order to get the digestive action of the saliva. With such care the patient may live fairly comfortably for from one to three years. Inevitably gastrostomy will be necessary in time. It is important to do this before the patient is moribund.

Irradiation by the Coutard technic influences the rate of growth. "Used early, it has an effect of arresting growth; used late, the toxemia is so great that the patient does not seem to recover from it and the end is hastened." Finally, it is most important not to make an erroneous diagnosis of cancer of the esophagus on account of the great additional mental anguish to the patient and his relatives.

Esophageal diverticula are not recognized as often or as early as they should be. The symptoms of pressure or pulsion diverticulum are quite characteristic and a patient who has suffered from them can readily make the diagnosis in others. These are first noticed usually in late middle life, although they probably all arise from a congenital weakness in that area where the primitive foregut fuses with the buccal stoma. The point on the posterior wall directly behind the cricoid cartilage is thus not covered with intrinsic muscle, and it is at this narrowest point of the alimentary tract that, due to an unusual congenital weakness, the act of swallowing results in a diverticulum. It usually extends to the left, and size of the pouch determines the severity of the symptoms. In the early stages there is usually a sense of irritation or of foreign material in the throat and perhaps an increase of mucus. Then particles of food lodge in the sac, causing enlargement, and a sense of obstruction high in the esophagus.

The enlarged sac with the weight of its contents sometimes causes actual obstruction; the loss of as much as 63 pounds from this, with corresponding loss of strength, has been reported. In such cases feeding through a catheter which has been introduced over a guiding thread is necessary to get the patient in condition to undergo operation. Cough from pressure on the recurrent laryngeal nerve is frequent. Regurgitation of food taken at the first part of the meal, or mucus, may be most annoying; to avoid regurgitation at night some patients have learned to wash out the diverticulum before retiring. Fairly early in its development there may be a characteristic noise on swallowing fluids. Pain in the neck is a rare symptom, and palpable swelling when the sac is filled with food is not constant. But just after the patient is given a drink of water, sudden pressure over the diverticulum forces the water back into the mouth and produces the same type of gurgling noise that is made on drinking in the conventional manner. The x-ray will confirm the diagnosis.

Traction diverticulum, usually at the bifurcation of the trachea results from contracture of inflammatory tissue in the adjoining structures. It causes few symptoms, and is rarely diagnosed before necropsy.

The proper treatment of pressure diverticulum results from the cooperation of the esophagoscopist with the general surgeon. With esophagoscope in place, the dissection of the diverticulum from without, with care to get the whole sac and yet not to remove part of the esophagus, can be readily accomplished in one operation. It is, of course, essential that the opening should be sewed up most securely to permit the passage of neither fluid nor air, and the patient is fed through a nasal tube for some ten days.

Though the term cardiospasm is perhaps not an ideal one, and "achalasia" and "preentriculosis" have been suggested as substitutes, in the present state of our ignorance on the subject, I shall continue to use the term which is so generally understood. There seems little doubt that there is a functional factor. The more important diagnostic symptom of cardiospasm is usually the history of regurgitation and food which has not been thoroughly acidified, although the patient usually believes he is vomiting. Sometimes there is a feeling of heaviness or pain in the chest after eating. The x-ray is of great assistance in the diagnosis, giving the typical dilated esophagus with the smooth generally conical termination at, just above, or just below, the apparent level of the diaphragm. However, mucus or retained food may give a roughened appearance, suggesting carcinoma, and sometimes the scar of a peptic ulcer will simulate cardiospasm. The treatment is dilatation of the cardia with a rubber bag under direct vision, and this operation often has to be repeated. It is my practice to make the first dilatation under a general anesthetic and the immediate improvement is remarkable. It is well for the patient to remain on a bland diet, avoiding ice cream and iced drinks, and unquestionably everything possible should be done to produce psychic quietude.

Congenital anomalies of the esophagus has been recognized for many years. The most common and the most serious form is that characterized by the upper portion of the esophagus opening into the posterior wall of the trachea, and it is always fatal during the first few days of life. In 1931 Findlay and Kelly called attention, with the report of nine cases, to a congenital shortening of the esophagus with a portion of the stomach above the level of the diaphragm. Last year Clerf and Manges added nine more cases. This condition may be recognized in childhood or in late middle life. Although dysphagia may not be recognized, this symptom is invariably present

and is often accompanied by distress after eating. This may vary from flatulence to severe epigastric pain. The patient often finds it necessary to take large quantities of fluid when eating to aid in swallowing. Regurgitation during meal time is not uncommon. Sometimes the diagnosis is made on account of a bolus of food lodging at the stricture.

Careful roentgenologic examination with the patient in the right-oblique-prone posture is the most important single point in the diagnosis. Esophagoscopy relieves dilation of the upper thoracic esophagus with a chronic esophagitis. "Unlike a normal hiatus, the narrowings found at the lower end of the esophagus offer marked resistance to the tip of the scope, suggesting an organic stenosis." In cases of organic stenosis esophagoscopy with bouginage is promptly followed by improvement, particularly in children. "The patients with ulceration at the junction of the esophagus and stomach present a difficult problem in treatment, pain being the most prominent symptom. Various combinations of alkalies in solution and bismuth subnitrate, dry on the tongue, afford temporary relief. Topical applications of nitrate, 10 percent, to the ulcerations seem to be helpful."

#### SUMMARY

A patient who complains of difficulty in swallowing, no matter how neurotic in appearance, deserves a thorough diagnostic study. This should begin with a case history, paying particular attention to the possible ingestion of a caustic. A general examination with proper x-ray studies, and investigation as to the possibility of syphilis and tuberculosis, should come next. This should be followed by a careful roentgenologic examination of the esophagus. Unless the diagnosis has already been established, esophagoscopy should then be made, and one should never hesitate to perform a biopsy on any tissue which suggests the possibility of cancer. If the esophageal obstruction is marked, or if it dates from the recent ingestion of a caustic, gastrostomy with retrograde dilatation by bougies for a time, with latter bouginage from above, is indicated. In the event that cancer develops, palliative treatment may give the patient from one to three years of fairly comfortable life even though the diagnosis is made late. If made early, esophagectomy is to be considered.

Pulsion diverticula may be ablated with comparatively little risk and with great relief to the patient.

Hiatal hernia and cardiospasm may be relieved by dilatation, though repetition may be necessary later.



# UNUSUAL CASES OF CICATRICIAL STRICTURE OF THE ESOPHAGUS

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The most common cause of cicatricial stricture of the esophagus is the swallowing of chemical escharotics. Commercial lye is usually the offender, but acid burns of the esophagus are occasionally encountered.

It is noteworthy that these burns from swallowing lye or acids are rarely complicated by obstructive laryngeal dyspnea requiring tracheotomy, despite the fact that the victims are frequently of the tender age when laryngeal obstruction is most easily produced. Hill<sup>1</sup> reported such a case which resulted fatally in an infant that swallowed washing powder. Case 1 is a report of a patient of mine that required tracheotomy for laryngeal edema following the ingestion of sulphuric acid.

## REPORT OF CASES

*Case 1.*—A. J., a white male child, aged two and one-half years, was admitted to Touro Infirmary January 4, 1933. He had swallowed an unknown quantity of sulphuric acid about seven hours earlier at his home in Picayune, Miss. The acid (automobile battery acid) had been left in a milk bottle on a porch, where the child found it.

On admission to the hospital his temperature was 103, pulse 200 and respiration 42. There was an ashy gray pallor of the skin and marked obstructive laryngeal dyspnea. There were acid burns of the lips, chin, left eyebrow, fingers and the entire oral cavity. The throat was filled with thick mucus. After a low tracheotomy, which I did at midnight under local anesthesia, the pulse dropped to 114 and the child fell asleep.

Glucose and saline were given intraperitoneally. Mouth feeding was inadequate and gastrostomy was done January 6, 1933, by Dr. J. D. Rives. The tracheotomy tube was removed January 21, 1933. String swallowing was attempted but was unsuccessful, and January 21 retrograde esophagoscopy was done and a 12 F. Jackson steel stemmed filiform bougie was passed upward into the mouth, where a braided silk string was attached and the esophagus threaded by pulling the string down and out of the gastrostomy with the bougie. The upper end of the string was then brought out through the nose and the two ends of the string tied together and looped over the ear, thus preparing the patient for the retrograde method of dilatation of esophageal strictures, according to the technic of Tucker.<sup>5</sup> Retrograde bouginage was begun at 12 F. and six months later had progressed to 32 F. Peroral esophagoscopy was not done until July 26, 1933, on account of the earlier feeble condition of the patient, and at that time showed a healthy esophageal mucosa with a stricture located 22 cm. from the upper incisor teeth. Retrograde bouginage was discontinued January 5, 1934, when the 40 F. bougie passed easily, and the

gastrostomy tube was removed. The gastric fistula would not completely heal due to a small prolapse of gastric mucosa and a plastic closure was done by Doctor Rives July 19, 1934. Esophagoscopy done at this time revealed a lumen so large that the location of the stricture was difficult to determine.

Cicatricial stricture of the esophagus is a rare complication of scarlatina. Vinson<sup>7</sup> reports two cases, one of recent origin in a child of eight years, and the other in a woman of thirty-eight years who had had recurring attacks of dysphagia since scarlet fever thirty-one years prior to his examination. Following is the history of a similar case which came under my observation:

*Case 2.*—Mrs. L. D. L., a white female, aged twenty-five, had had scarlet fever complicated by abscesses in the neck, at the age of fifteen years. Three months after this illness she began to have difficulty in swallowing, and from that time her food had to be finely divided for her to be able to swallow it. She at times resorted to massage of the neck in the suprasternal region to assist swallowing. Esophagoscopy revealed a 14 F. cicatricial stricture of the esophagus about 4 cm. below the cricopharyngeus. Peroral esophagoscopy bouginage was done on four occasions, but was discontinued because of lack of progress and dislike of the patient for the procedure. Gastrostomy was then done by Doctor Faust, June 20, 1928. Retrograde bouginage was begun nineteen days later with the Tucker 14 F. bougie and had been carried to 32 F., the largest size of retrograde bougie then manufactured, about six months later. The gastric fistula was allowed to close and bouginage was continued up to 45 F. by allowing the patient to swallow, under supervision, long woven silk bougies. Two years after dilatation was discontinued in this case, a 9 mm. full lumen esophagoscope was passed to the stomach with no resistance whatever at the stricture level.

Cancer may develop at the site of an old cicatricial stricture of the esophagus. In these cases two types of precancerous lesions may be noted—the cicatrix itself and the static esophagitis resulting from esophageal stasis in small lumen strictures. Magnusson<sup>3</sup> has reported a case of carcinoma of the esophagus in a man forty-one years old, in which case the tumor was located in the neighborhood of a cicatrix from lye swallowed at the age of eighteen months. Myerson<sup>4</sup> has reported carcinoma of the esophagus in a man twenty-nine years old, growing at the site of a cicatrix resulting from swallowing lye at the age of three years. The clinical appearance of the lesion in this case did not definitely suggest malignancy but the microscopic diagnosis was epithelioma.

A patient observed by me developed carcinoma of the esophagus at the site of a cicatrix from lye burn forty-one years after swallowing lye. His history follows:

*Case 3.*—H. R., a colored male, aged forty-five years, had had much difficulty in swallowing for two weeks and had lost much weight. He swallowed lye at two years of age and for five years after that was limited to semisolid diet; after that he ate everything, but occasionally had some difficulty in swal-

lowing. Skiagraphic examination showed a very much dilated esophagus in its upper third with a constriction approximately at the aortic arch. At esophagoscopy a slightly bleeding fungating, infiltrated area was found on the anterior wall of the esophagus slightly above the level of the aortic arch. Clinically the lesion was malignant and biopsy showed squamous cell carcinoma.

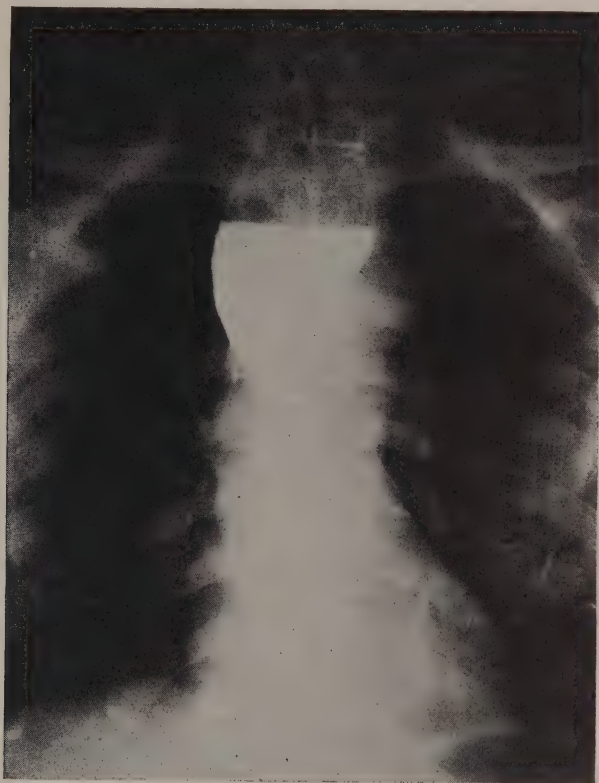


FIG. 1. Case 3. In which carcinoma developed at the site of an esophageal stricture forty-one years after the patient swallowed lye.

The esophagosopic findings in this case confirm Tucker's<sup>6</sup> conclusion that the final diagnosis in "stricture" of the esophagus should be made by roentgen ray study and direct esophagosopic inspection. Jackson<sup>2</sup> emphasizes the folly of attempting to make a diagnosis of esophageal disease without looking at the esophagus.

Treatment of esophageal stricture should be promptly carried out by someone trained in the management of these cases. The malnutrition of these neglected and sometimes mistreated little unfortunates, who have long been fed on a totally inadequately liquid diet, is pitiable. The following history is an example:

*Case 4.*—M. S., a white female child, aged seven years, developed an esophageal stricture after swallowing lye February 17, 1929. For one year unsuccessful

ful attempts were made by her physician to dilate the stricture by peroral string guided bouginage. She was then taken to a chiropractor, who treated her for two years by rubbing the stomach and the throat. She then again consulted a physician, gastrostomy was done and retrograde dilatation was carried on for two years to 24 F. According to the mother's story, no attempt was made to go beyond that point, as the attendant considered further dilatation too dangerous.

She came under my observation February 16, 1934, a stunted little seven-year-old girl, weighing thirty pounds. Fluoroscopic examination of the esophagus showed a stricture just below the cricopharyngeus. Retrograde bouginage was started at 22 F., and November 5, 1934, had progressed to 40 F. This five-year-old cicatrix was the most obstinate, unyielding, cicatricial stricture of the esophagus that I have encountered, but the result has been entirely satisfactory and the little patient now eats everything in an ordinary diet and has gained about ten pounds in weight. The gastrostomy tube was removed January 7, 1935, to allow the gastric fistula to close.

### SUMMARY

An account has been presented of the following cases:

1. A case of a child who swallowed sulphuric acid and required tracheotomy for obstructive laryngeal dyspnea, gastrostomy for feeding and retrograde esophageal bouginage for cicatricial stricture of the esophagus.
2. A case of cicatricial stricture of the esophagus resulting from scarlet fever.
3. A case of carcinoma developing at the site of an esophageal stricture forty-one years after the patient swallowed lye.
4. A case of lye stricture of the esophagus of five years' duration presenting unusual features.

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# EXOPHTHALMOS OF OBSCURE NASAL ORIGIN

(REPORT OF SIX CASES)

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While the classical picture of cellulitis or abscess of the orbit accompanied by frank suppuration in a neighboring sinus presents very little difficulty in diagnosis, there are times when profound disturbances in these tissues are caused by a sinus disorder which produces such meager symptoms and signs that its presence and significance may very readily be overlooked. One is impressed by the fact that vascular changes in the orbit at times reaching the extent of a marked exophthalmos with complete fixation of the globe, may often be brought about by a seemingly mild and insignificant sinus disease.

The extremely grave nature of orbital cellulitis makes it imperative that the cause of the edema be recognized early so that remedial measures, whether by local treatment or surgery, may be instituted without loss of valuable time; for it is only by this prompt action that many cases of orbital abscess with its attendant dangers are to be aborted.

The following cases, which have come under my observation during the past few years, illustrate some of the difficulties in diagnosis encountered in this condition and have been selected because of the fact that in them orbital changes were produced, the extent and gravity of which were out of all proportion to the seemingly mild and inconsequential nasal pathology.

No cases of orbital infection caused by frank suppuration in the sinuses have been included in the following report, but only those in which there existed a certain amount of doubt as to their nasal origin.

## REPORT OF CASES

*Case 1.*—White, male, three years of age, admitted to hospital March 16, 1928, with following history. Two weeks ago he had a severe cold with copious nasal discharge which lasted one week. Two days after all symptoms had subsided there appeared a watery discharge from the right eye. This increased in amount and swelling of the lids appeared during the next forty-eight hours. He was put to bed and local treatment to the eye and nose instituted. In spite of this he became progressively worse until, at the time of admission, the eye presented an alarming picture. There was marked swelling of both lids with chemosis of the bulbar con-

junctiva, the globe was protruding symmetrically with complete fixation, temperature 101 degrees, leucocyte count 18,600 urine negative. Pain was not a prominent symptom, nor was there evidence of great prostration. Examination of the nose showed moderately swollen turbinates on both sides which were readily shrunk down with a weak ephedrine and cocain solution. No nasal nor postnasal discharge was evident. X-ray examination showed a decided clouding of the antrum and anterior ethmoids on the right side.

Since appropriate local treatment had been carried out for several days without effect, the anterior ethmoids and antrum on the right side were opened intranasally under general anesthesia. The following day, after the removal of the packing, a sanguineous discharge appeared from the right nostril which was so profuse as to necessitate the constant cleansing of the lip. This was accompanied by a very noticeable amelioration of the orbital swelling and exophthalmos until, by the fifth day, the globe had assumed its normal position, and free movement in all directions was restored. In the meantime the nasal discharge had become quite thick, and at the end of ten days had entirely ceased.

During the six years subsequent to this attack he has had occasional severe colds without any recurrence of the orbital inflammation.

*Case 2.*—White, male, eight years of age, was examined in the home February 8, 1930. He had had a mild attack of grippe which was accompanied by a moderate amount of nasal discharge. For the past two or three days he had apparently been in perfect health with no discharge present. During the past twenty-four hours he had complained of a great deal of headache, pain in the left eye, and during the night the eye had swollen to such an extent that the lids could not be voluntarily moved.

At the time of examination the temperature was 101 degrees, there was marked swelling of both lids and conjunctiva, and the eyeball was pushed forward and toward the temporal region. No intra-ocular examination was made. The nasal mucosa was moderately inflamed with a very definite swelling of the middle and inferior turbinates on the left side. After thorough shrinkage some mucopurulent material was observed in the left middle meatus, and irrigation with saline solution produced a small quantity of mucus. Transillumination was clear on the right side, but the left antrum was hazy. Nasal treatment, consisting of shrinkage and irrigation, was carried out twice daily and by the third day the eye had regained its normal appearance and function.

These two cases were undoubtedly early orbital cellulitis, and most certainly would have gone on to abscess formation had effective nasal treatment not been started promptly.

*Case 3.*—White, female, 35 years of age, first seen March 9, 1930, at which time she had a peritonsillar abscess on the right side. This was opened and drained, and a normal convalescence ensued. A few days after recovery from the abscess she began to complain of a severe pain in and around both eyes, more particularly on the right side.

Nasal examination in the home revealed no abnormality. The turbinates were normal in appearance, there was no discharge present, and transillumination was exceptionally clear.

Twenty-four hours later the pain had increased and was accompanied by some photophobia and a noticeable swelling of the lids on the right side. After removal to the hospital, general examination, including neurologic, was essentially negative, temperature 99.4 degrees, pulse 88, respiration 20, leucocyte count 17,200.

Examination of the nose showed no signs of acute inflammation. The turbinates were normal in size and appearance, and the most searching and careful inspection revealed no mucopurulent material in the nose or nasopharynx. The olfactory fissure was clear, but a probe could not be passed into the sphenoid ostium. X-ray examination showed the sinuses to be entirely negative. By the following morning the condition had progressed and the left eye had also become involved. At this time an ophthalmologic examination showed a marked exophthalmos of both eyes with almost complete fixation of the globes. There was a decided photophobia and a moderate amount of edema of the lids. The bulbar conjunctiva was quite edematous and injected, the cornea appeared normal, the iris clear, and the pupils contracted. Tearing was fairly profuse, but no mucopurulent discharge was present.

Since she had had so recent a peritonsillar abscess, and since the nose seemed so unusually clear, we felt that we must be dealing with a cavernous sinus thrombosis, although no other signs than those mentioned could be made out. The following day the situation became rapidly worse. The edematous conjunctiva protruded from between the lids on both sides, the lids themselves were markedly edematous and injected, the globes were fixed, and pain was intense. The cornea was watched for any pressure changes and an external canthotomy considered. The fundi showed deeply congested veins almost black in appearance. No other changes were present. Twenty-four hour blood culture was negative, and her temperature had not gone above 99 degrees. In the meantime the search for nasal infection was continued. Twice daily the turbinates were shrunk down, suction applied, and the nose irrigated with saline solution. On the third day after admission our persistence was rewarded. While all previous irrigations had returned unusually clear without even a shred of mucus, on this day a large well-formed globe of clear mucus about 20 mm. in diameter came away in the solution, followed by several smaller pieces.

During the next twelve hours the change in her condition was truly dramatic. The pain was almost immediately relieved, the swelling of the lids and exophthalmos rapidly receded, so that by the following day there was a very noticeable improvement in the ocular symptoms. At each subsequent nasal treatment for several days a fair amount of mucopus was obtained from the postethmoid sphenoid region on both sides. The conjunctiva receded behind the lids and ocular movements were so rapidly restored that by the third day following the first evacuation of sinus discharge, free motion was established in all directions and the globe had assumed its normal position. After remaining in the hospital three more days for nasal treatment and observations, she was discharged symptom free. Some months later ophthalmologic examination showed  $\frac{20}{15}$  vision in each eye and since that time there has been no recurrence of nasal or ocular symptoms.

The very sudden and complete restoration of function immediately following the appearance of nasal discharge and the afebrile course of the disease led us to the conclusion that the startling changes in the orbit had been caused more by the blocking of orbital circulation from a practically symptomless sphenoid infection than from an orbital cellulitis caused by direct extension of infection.

*Case 4.*—White, male, age thirty-five. First examined in the office August 14, 1930. During the past six months he had had two severe colds during both of which there had been some inflammation of the eyes. About ten days ago he had another severe cold with a large amount of nasal congestion and discharge.

The discharge had practically ceased, but for about two or three days there had been some pain around the left eye. When he awakened this morning there was a marked swelling of the lids on the left side with profuse tearing. Examination by his ophthalmologist showed the bulbar conjunctiva to be very edematous; there was limitation of motion accompanied by a very definite protrusion of the globe outward and toward the temporal region, the pupil was dilated, and the retinal veins dark and full.

Examination of the nose revealed a very decided acute inflammation and swelling of the middle turbinates on both sides. Thorough shrinking and irrigation produced a large amount of mucopurulent material which seemed to come from the middle and superior meati on both sides. Transillumination was clear over the frontal region, but both antra were somewhat hazy. X-ray examination showed only a slight clouding in the left frontal.

Vigorous local treatment was carried out twice daily for three days, at the end of which time, the swelling and exophthalmos had entirely disappeared. At the end of one week the sinuses appeared entirely clear.

The interesting feature of this case is that, during the four years since the original attack, this same patient has had the same condition in either one or both eyes at seven different times. The left side has been more frequently involved than the right. On each occasion, it has responded very quickly to local treatment and in most instances he has pursued his work with practically no interruption during the course of the attacks.

It would seem quite probable that in this case there is a bony dehiscence which facilitates the transmission of nasal inflammation to the orbital tissues, but he has refused any surgical procedure for permanent relief of the condition.

*Case 5.*—White, female, sixty years of age, examined in my office December 3, 1931. Two weeks before she had noticed some swelling in the left eye. Ophthalmologic examination showed a moderate edema of the lids with widening of the palpebral fissure. Exophthalmometer reading, right eye 18, left eye 24. The fundus was normal. This condition remained present with slight variations for a week, at which time the exophthalmometer reading was right eye 22, left eye 30. There was no history of any recent cold. She had not noticed any nasal obstruction, nasal or postnasal discharge. The only symptom elicited was that of a mild sore throat which she had had previous to the attack.

Nasal examination showed a very narrow nose with both middle turbinates impinged on the septum. There seemed to be a distinct inflammation and swelling of both middle turbinates. After thorough shrinking, a probe could be passed with difficulty between the middle turbinates and septum on the left side, but the sphenoid ostium could not be located. X-ray examination showed a very slight thickening of the lining mucosa of both antra. The nose was packed with argyrol tampons, after which suction was applied and the nose irrigated with saline solution. A few pieces of mucus were obtained. The following day she felt that there had been a decided improvement in the eye. The nose was treated daily for several days and on each occasion some mucus was washed out. In the meantime she had been getting a very active postnasal discharge and at the end of the fifth day all swelling had disappeared. The exophthalmometer reading was right eye 21, left eye 22. She was then treated at less frequent intervals until the postnasal discharge had entirely cleared.

During the three years since the original attack she has been seen two or three times each year with an acute rhinitis. Only on one occasion has there been



any recurrence of the orbital symptoms. This consisted of a very slight swelling of the lids with no exophthalmos and cleared up within forty-eight hours under treatment.

*Case 6.*—White, female, age thirty-eight, first seen November 21, 1933. Four days before she had noticed some swelling around both eyes, more particularly the left, until at the time of examination there was a marked edema of the conjunctiva present. She complained of no pain except when the eyes were rotated. There was a very decided proptosis slightly toward the temporal region, and a very definite diplopia in the upper field. Vision  $20/15$  in each eye. Fundi appeared normal. Movements of the left globe were limited in all directions. Examination of the nose showed a moderate amount of congestion in both middle turbinates. After thorough shrinkage, no discharge could be made out. X-ray examination at this time showed clouding over both antra and both anterior ethmoids, but the involvement seemed to be more on the right than the left. The frontals and posterior sinuses were clear. Transillumination showed slight haziness over both antra. The left antrum was punctured and irrigated, the solution returning practically clear.

She was sent to the hospital for nasal treatment which consisted of thorough shrinking, argyrol packs, and irrigation. For forty-eight hours there was no improvement in the condition, and we were on the point of advising an intranasal ethmoidectomy and antral window when some mucopurulent material began to be discharged. There was immediate improvement in the orbital condition and in about four days the eye had attained its normal position with restoration of free movement. The sinus condition was rather obstinate and persisted for several weeks. Since that time she has had two or three colds but has had no recurrence of the sinus or orbital involvement.

### SUMMARY AND CONCLUSIONS

It would seem that from consideration of this admittedly small group of cases a few interesting observations may be made:

1. In any case of orbital edema careful and repeated examinations of the sinuses should be made and every resource at our command used before concluding that the sinuses are not the causative factor. Perseverance may bring to light an obscure and apparently insignificant infection.

2. There may be a complete absence of any history of recent nasal inflammation, the condition being so slight as to entirely escape the notice of even an intelligent patient.

3. Obscure lesions in the postethmoid sphenoid region may produce circulatory disturbances in the orbit which closely resemble cavernous sinus thrombosis.

4. Because in each instance improvement became apparent within a very short time after the institution of effective nasal treatment, it seems certain that, if seen and diagnosed before the formation of an orbital abscess, a large proportion of these cases may be permanently relieved by local treatment or minor intranasal procedures.

MEETING OF THE WESTERN SECTION HELD IN LOS ANGELES, CALIFORNIA, ON JANUARY 18 AND 19, 1935, UNDER THE CHAIRMANSHIP OF DR. FRANK A. BURTON OF SAN DIEGO, CALIFORNIA.

HERPES ZOSTER OTICUS

(WITH REPORT OF TWO CASES)

By HILL HASTINGS, M.D.

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I wish to present these cases because they are rare, and also, because I am convinced that many of us at times have failed to make correct diagnoses.

In looking through the Transactions of our Society I could find only one report of herpes zoster oticus and that was made by Vail twenty-nine years ago. However, there have been a few papers on this subject in the medical journals. It is not my intention at this time to go beyond reporting these cases as follows.

*Case No. 1.*—Mrs. I. M. L., age eighty, had been deaf for many years; was using an electrical aid for hearing; had been treated off and on for two or three years without benefit. Her hearing test prior to the onset of the attack which I am now reporting, showed nerve deafness in each ear. The deafness in the right ear was almost complete and there was little hearing function in the left ear. She had never had any dizziness or pain.

On October 27, 1933, she returned because of the sudden onset, occurring a week previously, of pain in the right ear, dizziness and a bloody discharge without any acute respiratory infection, and without any other apparent cause. The dizziness at the onset of this sudden attack was so severe as to keep her in bed. It had gradually grown less severe, and before her coming to my office had almost subsided. Examination showed a blood blister on the anterior wall of the right external auditory canal that prevented a view of the fundus. On pricking this blister and wiping the bloody serum, a similar blister was found on the anterior part of the drum membrane. The posterior part of the membrane, after drying carefully, was seen to be unaffected. There was no fever, no facial paralysis, no mastoid tenderness and no nystagmus. Two days later the right side of the chin and lip became swollen and red with small vesicles in the vicinity of the mental foramen. The occurrence of the herpes of the chin and lip on the same side as the acute ear trouble brought to my memory a paper written by J. Ramsey Hunt, the neurologist, in 1907 in the *Journal of Nervous and Mental Diseases*, in which he made a lengthy report on "Herpetic Inflammation of the Geniculate Ganglion; a New Syndrome and Its Complications." I found that in 1910 in the *Archives of Internal Medicine*,

Hunt made an exhaustive report of the symptom-complex due to inflammation of the ganglia of the seventh, eighth, ninth and tenth nerves, with reports of cases where herpes of the auricle, ear canal, drum membrane, face, tongue and throat were found. Hunt thereby explained the cause of symptoms and signs in our specialty that he said had evidently been overlooked by many of us. In further looking up references of herpes oticus I determined that this patient of mine had had an attack of inflammation of the ganglion of the fifth nerve, and that the pain and bloody serum from vesicles of the auditory canal were not due to a local inflammation of the drum membrane or the ear canal nor were the vesicles of the chin in the region of the inferior mental foramen due to local causes, but both ear and chin symptoms and signs were due to involvement of the ganglion of the fifth nerve. It was the first case in my practice that I had recognized. It is likely that other patients whom I have have suffered from the same cause but, unable to see them during the vesicular stage, I did not make a correct diagnosis.

*Case No. 2.*—W. F. B., male, seventy-seven years of age, was admitted to the Los Angeles General Hospital October 2, 1934, with the following complaint: Seven days previous he had been seized with severe pain in the right ear, lancinating in type, and severe dizziness. The dizziness was so severe he said he had to get down on the floor and crawl to his bed where he stayed for several days until the dizziness became less. He also noticed that he was suddenly deaf in the right ear, and had severe pain in the right side of the chin, mandibular region and the preauricular region. Vesicular eruption appeared two days later on the chin and in front of the right ear. Five days after the above symptoms and signs commenced a complete right facial paralysis developed. On the day of admission to the hospital, just seven days after the onset of the trouble, tests showed normal hearing in the left ear and almost complete loss of hearing in the right ear. The patient said when lying on the left side he could not hear the patient in the next bed speak. The vesicles in the preauricular region on the right side of the jaw had evidently ruptured and were healing. Examination of the right ear canal and drum membrane showed a large ruptured vesicle on the anteroinferior portion of the canal, and the canal was filled with a serosanguinous discharge. After cleaning out this discharge the drum membrane was seen to be red on the posterosuperior portion but there was no vesicle on the drum membrane. The remaining portion of the drum membrane was only slightly inflamed. There was a complete right facial paralysis, and the temporal and preauricular regions were extremely tender to touch. The patient had to lie still and not move from side to side or sit up, because any movement produced a very disturbing dizziness. Patient stated the dizziness, however, was not nearly as bad as when his ear "broke" on October 1, and that the hearing had become somewhat better. The eyes reacted normally to light and accommodation, the pupils were equal and regular. The Wassermann test was negative. Two days later the pain had decreased and his hearing was somewhat better. The eruptions on the face were healing quite rapidly. Eleven days later the vesicles were almost entirely healed, and he could hear conversational voice eight feet in the right ear. The facial paralysis seemed to be slightly less. The right ear canal and drum membrane were practically normal. Vestibular tests showed no reaction in the right semicircular canals with the head in any position. The douching was done with

water at 68 degrees F. Patient was discharged from the hospital October 26, twenty-four days after his entrance. At that time there was noticeable improvement in the facial paralysis and the hearing had improved. The skin of the right side of the face was clear and the dizziness had entirely subsided.

I should add, the diagnosis on entering the hospital was quite uncertain. Dr. A. H. Miller, Senior Resident in the Ear, Nose and Throat Department, Dr. Cyril Francis and Doctor Fitzgibbon of the medical resident staff began to study the literature and suspected that it was a case of Ramsey Hunt ganglion syndrome. This diagnosis was confirmed by Dr. J. M. Nielson and Dr. Samuel Ingham, attending neurologists of the hospital. I was told about the case and when I examined the patient it seemed quite clear that it was another one of these rare cases, where the signs and symptoms were due to inflammation of the cranial ganglia. In this case the ganglion of the fifth nerve was involved, and also the geniculate ganglion of the seventh nerve, which latter ganglion was not involved in my first case.

My last examination of this patient was made in my office on November 21, 1934, at which time his facial paralysis had almost subsided and the eruptions had disappeared but small scars were still present in the preauricular region. The hearing had become almost normal.

The vestibular tests at the time of my last examination were as follows: No past pointing with either arm. No spontaneous nystagmus. Douching the right ear canal with water 66 degrees showed no nystagmus after six minutes and no past pointing. The patient was made slightly dizzy after three minutes douching. Douching the left ear produced slight dizziness at a minute and a half, but no nystagmus or past pointing. The falling reaction was normal on turning five times in ten seconds to the right, and also when turning five times in ten seconds to the left. The patient said he was never dizzy now except when looking quickly up or down and particularly when stooping over. His hearing tests showed: equal hearing in the two ears (twelve feet plus, which the patient said was as good as he has heard for several years). The Weber was not lateralized. Bone conduction for C 256 fork was equal on the two sides; Rinne was plus in each ear; the lower tone, C 64 fork, was normal in each ear; the upper tone, C 2,046 fork, was especially good for his age—minus ten seconds in each ear.



# AN EVALUATION OF RADIATION TREATMENT FOR CARCINOMA OF THE LARYNX

(WITH SPECIAL REFERENCE TO THE COUTARD METHOD)

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Roentgen discovered the x-ray in 1895. Its use in therapy may be divided into three periods—optimistic, pessimistic and realistic. The first era extended to about 1906 and was associated with a paucity of scientific and clinical knowledge, inadequate facilities, empiricism and innumerable accounts of the remarkable cure of intractable dangerous diseases.

The failure to substantiate these early therapeutic reports, the injurious effects observed as time went on, and the fact that there was no satisfactory method of estimating or controlling the quantity and quality of radiation administered, resulted in a period of pessimism, which endured to about 1912. Then came the third period, dominated by physicists, biologists and scientifically inclined physicians, whose efforts have developed reliable apparatus and instruments for the production and measurement of x-radiation.

While an occasional good therapeutic result was obtained, roentgen therapy up to 1920 was hampered by lack of efficient voltage and inaccurate methods of estimating dosage. In 1920, Seitz and Wintz advocated the massive dose technic. By this method as large a dose as possible was administered in the shortest possible time and the term "cancer dose" came into being. It soon fell into the discard. Regaud studied the influence of radiation upon cells during the process of division and suggested the technic by which the treatment by comparatively small intensities is extended over a very long period so that more neoplastic cells are exposed to radiation during the phases of mitosis and when most sensitive to the destructive influences. The saturation method of Pfahler is a modification of Regaud's technic. According to this method, an attempt is made to keep the diseased tissues saturated with radiation over a comparatively long time during cell division by doses carefully calculated as to per diem loss and normal tissue tolerance, based upon the latent period of effects until ultimately a maximum dose is delivered, which is comparatively larger than can be safely administered by other procedures.

The application of irradiation to malignant disease of the larynx presents problems peculiar to this region. Most malignant neoplasms of this region (96 percent) are of the squamous cell variety and generally of the adult type cell, which is particularly radio-resistant.

Anatomically, the larynx is poorly adapted to withstand insult from radiation. The cartilaginous box with its relatively thin soft tissue lining bears the brunt of injury due to the irradiation. Then, in addition, pyogenic bacteria are constant inhabitants of this region. This organ is in constant function, unless there has been a tracheotomy. These factors combined, present a situation that makes the successful treatment of laryngeal cancer a difficult achievement. It is little wonder that the older methods so signally failed in the performance of good results. In fact, irradiation usually complicated the condition and contributed only to the unfortunate victim's suffering.

The splendid record that has been achieved by surgery in the treatment of intrinsic cancer of the larynx has influenced laryngologists generally in the opinion that laryngofissure or laryngectomy should be employed. Extrinsic cancer, whether primarily so or from extension from an intrinsic lesion, has rarely been cured by surgery or, for that matter, by formerly used methods of irradiation.

The rôle of x-ray and radium irradiation in laryngeal cancer has been that of an adjunct to surgery, being used before or after operation to prevent glandular invasion, or in those cases which were not amenable to surgery because of the location alone and the bad results from that form of therapy were all too frequent. Many patients with laryngeal cancer were lured away by the false promises of irradiation till beyond the point of surgical aid.

Coutard, in 1920, began treating cancer of these parts with a new method of irradiation. In 1931 he reported his results which started the radiological and laryngological worlds. The results he gave concerned patients from five to ten years after treatment. Of seventy-seven patients with laryngeal cancer, twenty-two (28 percent) were symptom free.

The technic developed by Coutard consists of the use of high voltage x-ray (200) KV. with protracted fractional dosage. It employs a heavy filtration and by giving two doses each day, over a very long period of time (three weeks to forty days), a very great intensity can be administered. It differs from the saturation method because it permits a greater total dose. In all previous methods the limit of tolerance has been the skin erythema (and radiologists have been timorous in exceeding this effect). Coutard

carries the toleration limit far beyond the erythema dose to actual vesicular dermatitis.

For the more technical details I will refer you to Coutard's reports as well as the works of American radiologists.<sup>1,2,3</sup>

A description of the clinical course of the patient undergoing treatment by the Coutard method is in order because of the dramatic phases which may frighten one if they are not well known. It has been determined that one may push the dosage to the point of producing an intense vesicular dermatitis with the prospect of complete recovery of normal structure from the insult. The mucous surfaces are more vulnerable and the so-called radio mucositis begins before the reaction in the skin. The first reaction to be noted is a ground-glass appearance which begins at the union of the pillars and uvula on about the tenth day. By the thirteenth day this area is covered with a grayish false membrane. By the fourteenth or fifteenth day the posterior wall of the hypopharynx is likewise affected. The epiglottis, interarytenoid region and base of the tongue show membrane about the eighteenth day. The rest of the larynx is covered by the twenty-first to twenty-second day. The skin shows its reaction first by an ever increasing amount of tanning. This continues until about the twenty-sixth to twenty-eighth day when vesiculation begins, followed by a loss of the whole epidermal layer. Healing begins at once and, in two or three weeks more, the surface is again covered with epidermal tissue.

Edema of the various salivary glands during the course of treatment is common. Edema of the mucous membrane and deeper tissues may so close the airway as to necessitate a tracheotomy. Dryness of the mouth, thickening of saliva, loss of sense of taste and dysphagia all occur and are annoying to the patient. Chronic edemas becoming harder and harder as time goes on, varying in intensity as more or less blood comes to the area, are due to impedance of return circulation by blood vessel changes and destruction. Chondritis, tissue necrosis, rupture of blood vessels, muscular atrophies and paralyses are all mentioned, but occur with much less frequency than with any former method.

Most patients become weak, lose about twenty pounds, are listless and often require hospital care for several days at the height of their reaction. During this period frequent mouth irrigations, the use of nasal feedings because of dysphagia and mild sedatives go far to relieve the worst of the suffering.

The upset to the general well-being of the patient subjected to this form of therapy is considerable and a patient who is not in fairly

good general health is in nowise a good risk to survive the effects of the treatment itself. It is our conception that a patient must be able to withstand more than he would if he were subjected to laryngofissure, but hardly that which he would have to withstand were he to have a total laryngectomy.

To date we have observed sixteen cases of laryngeal cancer treated by Coutard's method. Of these, three have died of general exhaustion and inanition. One died of laryngeal hemorrhage. One case remains unchanged. In the rest of the series a very definite change occurred. The local lesions disappeared. Our oldest case stands but one and a half years since the beginning of treatment so that we cannot count any as definitely cured. Tissue changes in surrounding mucosa and skin are present in all cases. The skin is hairless, with a smooth glistening surface. There is a leathery feel with some subcutaneous brawny thickening. Small areas of telangiectasis are present. The mucous membrane appears paler and thicker than normal. Usually there is some thickening of the site of the original lesion, often accompanied by submucous edema. These areas of local thickening are being anxiously watched for evidence of recurrence. In case No. 7, an incision was made over the swollen arytenoid region and a deep specimen was removed for biopsy. This showed no cancer cells.

Two cases that died, No. 14 and No. 16, were examined post-mortem. Histological examination indicated that the cancer had been completely destroyed in both. Twelve are alive and in good condition, with no objective evidence of the neoplasm.

Coutard's treatment offers a definite hope of cure in extrinsic laryngeal cancer and is the treatment of choice to date. Whether or not its use in intrinsic cancer will replace surgical treatment can be answered only by the test of time.

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Name	Lesion	Duration	Treatment	Result
N. R., age 49, male.	1-6-34. Squamous celled carcinoma right cord at vocal process.	Hoarseness, 2 months.	6025 R 1-12-34 to 3-2-34.	9-6-34. Good voice, slight thickening, mm., of larynx. Leathery skin. Good health.
I.	Grade III.			
M. J. Q., age 62, male.	10-30-33. Grade III. Squamous celled epithelioma.	Hoarseness, 2 months.	6030 R 11-13-34 to 12-8-33.	3-14-34. Arytenoids thick. Cords moving. Good voice. Edema epiglottis. Rt. arytenoid enlarged. Weight normal.
II.	Extensive involvement right cord and right aryepiglottic fold. Extension into vallecula and extension over cricoid.			
R. S., age 45, male.	Dirty gray ulcer. Posterior half left cord.	Hoarseness began 6-1-33.	6060 R 2-5-34.	5-6-34. Died of inanition.
III.	1-29-34. Squamous celled carcinoma. Grade I.	Treated for lues, Wassermann neg. Tracheotomy. 1-12-34.	2-23-34.	
E. M. V., age 67, female.	Squamous celled carcinoma. Grade II. Rt. arytenoid and pyriform sinus.	Hoarseness, 3 months.	6483 R	Died 11-30-34.
IV.				
C. C. K., age 62, male.	11-2-33. Inflammatory lesion. Keratotic epithelium. Hyperkeratosis of larynx of long duration. Oct. 2-34. Edema and fixation of rt. arytenoid. General swelling both cords. White patches on left cord.	Under observation for ten years for pachydermia larynx. 10-2-34. Throat painful and hoarseness for several months. Malignant transition evident but not proven by biopsy.	6030 R 11-13-33. 12-8-33.	When last seen there was no essential change in nature of lesion.
V.				
J. J. H., age 60, male.	Squamous celled carcinoma. Grade IV. Large mass attached to left aryepiglottic fold and pyriform sinus filling glottis.	Hoarseness, 6 weeks. Lost weight, 20 lbs. in 5 months.	3382.5 R 7-7-33 to 9-21-33.	1-11-35 "Cure." Slight limitation of left cord movement. No evidence of lesion. Voice good.
VI.				
S. S., male.	7-9-34. Squamous cell epithelioma. Grade II. Mass projecting into hypopharynx from right pyriform sinus and epiglottis region.	Hoarseness, 5 years. Difficulty in breathing for 3 months. Difficulty in swallowing for 2 months.	5175 R 7-27-34 to 8-6-34. 6-18-34.	11-17-34. Surface larynx smooth, thickened both sides, more on left. Aryepiglottic fold, rt. cord seen. Left cord partially fixed. Radon implants. Left arytenoid.
VII.				
C. V., age 50, male.	Squamous cell carcinoma. Grade III. Ulcerated granular mass involving rt. ventricle ventricular bank and lateral wall.	Pain in throat for 2 months. Hoarseness, 2 weeks. Hemoptysis, 4 weeks. 3-8-32.	Laryngo fissure, 3-23-33. May, 1932. Recurrence. 5-18-32 to 6-18-34. Six courses x-ray. Neck, 7500 R Supraclavicular, 11900 R Mediastinal, 9000 R Total, 28000 R	12-6-34. General condition good. Left arytenoid still swollen, right cord moves freely. No activity in neck. Skin deeply pigmented, and moderately fibrosed.
VIII.				
R. McC., age 58, male.	Epidermoid carcinoma, moderately malignant. Nodular ulcerated lesion involving left side of epiglottis, left vallecula and left aryepiglottic fold.	9-1-34. Choking, 10 weeks. Swelling left neck, 10 weeks. Hemoptysis, 3 weeks. Loss, 22 lbs. Difficulty swallowing.	11-6-34 to 11-24-34. 4800 R	December 28, 1934. Good condition, swallowing with ease. Mucous membrane of pharynx and larynx edematous. No definite tumor seen.
IX.				

Name	Lesion	Duration	Treatment	Result
O. R., age 46, male.  X.	Rt. pyriform sinus filled with nodular ulcerated mass, also involving rt. arytenoid and extending up to base of tongue. Hard nodule at angle rt. jaw size of lemon, also above clavicle freely movable.	Dysphagia, 1 year. Hoarseness, 3 months. Swelling at angle of right jaw, 3 months. Loss, 35 lbs. in one year. Swelling above right clavicle, 2 months.	11-8-34. Began x-ray. 1157 R	12-20-34. Slight infiltration, right pyriform sinus, which is disappearing. Eats without difficulty. Fairly good voice. Rest of masses disappeared.
F. H., age 67, male.  XI.	Left pyriform sinus filled with infiltration with a granular surface. Involves left aryepiglottic fold. Arytenoid and pyriform sinus. 2-24-34. Very highly malignant epidermoid carcinoma.	Pain in throat, 3 months. Hoarseness, 3 months.	2-26-34. 3-20-34 6000 R	1-3-35. General condition excellent. Left pyriform sinus clear, with no evidence of disease. Both cords normal, move freely. Skin mottled.
H. C., age 72, male.  XII.	7-1-34. Quite highly malignant. Epidermoid carcinoma. Large nodular tumor mass of the left arytenoid obscuring left side of larynx. Moves on phonation. Saliva in left pyriform sinus.	Pain deep in throat, 3 months. Odynphagia, 3 months. Loss, 75 lbs., 6 months. Hoarseness, 1 month.	8-14-34 to 9-4-34 5400 R	12-20-34. General condition excellent. Tumor gone. Skin pigmented but soft. No fibrosis.
S. L., age 50, male.  XIII.	Epidermoid carcinoma. Rt. pyriform sinus filled with firm smooth nodular mass.	External incision in thyroid area for "abscess," 3 months. Hoarseness, 3 weeks. Hemoptysis, 2 weeks. Pain in throat, 3 weeks. Odynphagia, 3 weeks.	10-27-34 to 11-16-34. 5000 R	1-3-34. Excessive salivation. Marked injection of mucous membrane of pharynx. Epiglottis and arytenoids still swollen. No tumor seen although pyriform sinuses cannot be seen because of swelling. Skin deeply pigmented.
W. B., age 51, male.  XIV.	1-22-34. Epidermoid carcinoma. Grade III. 6-21-34. Epidermoid carcinoma. Grade I. 1-22-34. Malignant growth involving anterior one-half left vocal cord. 5-29-34. Entire glottis infiltrated. Left cord fixed. Percent.	Hoarseness, 6 months. Dyspnea, 4 months. Laryngo fissure. 1-25-34. Recurrence. Laryngo-fissure never healed.	7-2-34 to 7-25-34. 6000 R	Died. Terminal severe hemorrhage from superior thyroid artery. No cancer demonstrable on postmortem histological examination.
W. W., age 48, male.  XV.	2-6-34. Highly malignant squamous cell carcinoma. Tumor mass in anterior. Commissure extending back beneath cords. Rt. arytenoid fixed. Swelling left side larynx.	10-5-32. First thyrotomy. 2-6-34. Second thyrotomy, for recurrence with dyspnea.	2-16-34 to 3-14-34. 6900 R	Regained normal weight. Breathes and swallows normally. Hoarse voice. Marked thickening. Arytenoids and aryepiglottic folds.

<i>Name</i>	<i>Lesion</i>	<i>Duration</i>	<i>Treatment</i>	<i>Result</i>
W. A. S., age 63, male.	Large tumor right ary- epiglottic fold, ary- tenoid and pyriform sinus.	Dysphagia, 2 months. Dyspnea, 1 month.	4-21-34 to 5-9-34. 6757 R	4-21-34. Large part tumor re- moved by fulguration. 5-4-34
XVI.	Squamous celled carci- noma. Grade III.			Extensive sloughing. Tumor not seen. Rapidly down hill and died 9-24-34. Autopsy: Extensive nec- rosis of all soft tissues of larynx. Cartilages not involved. No malignant cells found.

# OBJECTIVE AND SUBJECTIVE TINNITUS AURIUM OF VASCULAR ORIGIN\*

(REPORT OF CASE PRESUMABLY DUE TO CEREBRAL ANEURYSM)

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A study of early literature discloses considerable skepticism as to the possibility of diagnosis of cerebral aneurysm during life. Sands<sup>1</sup> states that "the surrounding brain tissue ordinarily adjusts itself well to the slowly expanding growth; hence, their recognition before rupture is well nigh impossible." In the later literature, clinical studies have been reported by many authors which have established the fact that it is a pathologic condition that lends itself to a correct diagnosis during life.

According to Schmidt<sup>2</sup> who reports a group of twenty-three cases, the first known description of intracranial aneurysm was that of Biumi in Milan in 1765. Green<sup>3</sup> in a contribution on the subject in 1878 states that "Henning calls attention to the fact that brain murmurs were first investigated by Fisher of Boston in 1832."

## LOCATION, AGE AND SEX INCIDENCE

According to Gowers in an article written in 1893, these aneurysms are most frequently found on the middle cerebral artery, and thereafter with decreasing frequency on the basilar, internal carotid, anterior communicating, vertebral, posterior cerebral and the inferior cerebellar arteries. Von Hofmann<sup>4</sup> in an article in 1894 stated the order to be the middle cerebral, internal carotid, anterior communicating, basilar and the vertebral arteries, and the frequency of aneurysms on the basilar artery as being variously computed.

There also are differences of opinion as to the prevalence of cerebral aneurysm on one side or the other, the greater number having been placed on the left side in those case reports reviewed by the writer. Lebert<sup>5</sup> states that aneurysm of the internal carotid artery is believed by him to appear more often on the left side because of the fact that on this side the carotid artery arises directly from the aorta.

There also have been differences of opinion as to the incidence of cerebral aneurysm in the different sexes, the greater percentage, however, having been reported in women. According to Busse,<sup>6</sup>

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Wichern<sup>7</sup> and Schmidt, age is no factor in its occurrence, it frequently being found in individuals between the ages of thirty and sixty.

According to Beadles<sup>8</sup> in an exhaustive study of 555 cases, aneurysm of the internal carotid artery in its course through the sinus cavernosus caused symptoms in 69 percent of the cases studied. Symptoms such as paralysis of the eyes associated with neuralgia of the ophthalmic nerve, diminished vision and trigeminal symptoms along with tinnitus aurium, are manifested in these cases.

### OBJECTIVE TINNITUS AURIUM

Weil<sup>9</sup> states that there are two distinct types of tinnitus aurium easily distinguishable objectively that are the result of two distinct causative agents. The first type is characterized by a peculiar crackling noise not unlike the sound caused by the snapping of two finger nails together. It is caused by a clonic spasm of the muscles in and around the eustachian tube. The muscle most commonly affected is the tensor veli palati. In the second class, the sound perceived both objectively and subjectively is always a hum or murmur synchronous with the systolic heart sound, and is not an unusual concomitant of cerebral aneurysm. Kafka<sup>10</sup> describes this objective sound as one of a constant or intermittent nature which the examining physician himself can hear. He reports these sounds as being associated with vertigo at times and often being found in aneurysm of the carotid artery.

Poorten<sup>11</sup> believes that objective noises in the ear are usually the result of an aneurysm. Moos<sup>12</sup> reports a case in which persistent subjective noises existed so as to cause insanity of the patient, which case at autopsy showed the jugular bulb to be tremendously enlarged, and he attributes the tinnitus in this case as being due to the enlargement of the bulbus. Impairment of hearing is usually associated with tinnitus aurium in these cases.

Green in his early contribution to objective and subjective systolic murmurs in the ears states, "Poorten gives a résumé of other cases of objective murmurs which have been described. The first of these was stopped by compression of the arteria auricularis posterior and is attributed by Grüber<sup>13</sup> to an aneurysm of the basilar artery and by Von Troeltsch to peculiarities in the branches of the posterior auricular artery and changes in the parts to which this artery is distributed. In the second case there was a systolic murmur in the heart, ear and head which probably was a transmitted sound from the heart, and in the third case there existed an arteriovenous aneurysm between the

internal carotid artery and the cavernous sinus." Green divides these murmurs into three classes as follows: (1) physiological murmurs in the internal carotid artery, (a) dependent on partial stenosis of the carotid canal, (b) probably caused by a reduced vascular tension and dependent upon disturbances in the vasomotor system; and (2) pathological murmurs from aneurysms of the vessels of the head.

Iglauer<sup>14</sup> presented a case in which an audible tinnitus was present for more than ten years. The sounds disappeared when pressure was applied over the carotid artery and also when the head was rotated to the side of the lesion. In the case to be presented here, objective and subjective sounds are present and disappear upon pressure and rotation of the head as described in the case reported by Iglauer.

In the following case there is present an objective as well as subjective tinnitus presumably due to a cerebral aneurysm on the right side. We wish to express our appreciation to Dr. Cyril B. Courville for his assistance in correlating the neurological data.

#### CASE REPORT

*Personal History.*—The patient is a female, fifty-three years of age, married and a housewife. She had mumps and diphtheria as a child and after taking some serum for the latter developed an abscess of the abdominal wall. Some years ago she had a rather marked arthritis which disappeared after the extraction of teeth. There is quite a definite history of traumatic sphenopalatine neuritis which was undoubtedly caused by repeated intranasal surgery on the left ethmoid sinuses. Menstrual periods began at the age of eighteen and menopause was noted eight or ten years ago. The husband is living and well at the age of sixty. There are no children.

*Present Complaint.*—There is pain and tenderness beneath the left eye associated with irregular drainage from the nose. There is tinnitus and deafness in the right ear existent since April, 1934.

According to the patient's history and her belief, the trouble dated from 1926. At this time she was douching her nose with Zonite and she noted that the solution was apparently too strong since the mucous membranes felt burned. The solution was immediately washed out, but since that time she has had definite trouble with her nose. Sinus trouble dates from this period, with a gradual development of pain and tenderness in the region beneath the left eye and just adjacent to the nose. This pain has been relieved at various times by cocaineization of the sphenopalatine ganglion.

On two occasions during the past year she underwent intranasal exploration of the left ethmoid areas. During this interval she noticed that her vision was definitely blurred, and this occurs whenever there is not free drainage from the nose. Coincident with this nasal difficulty there have been recurrent attacks of pain in the back, which the patient designates as lumbago.

*Intracranial Symptoms.*—The first symptoms which seem to be definitely associated with some pathology within the skull were noticed nine months ago. At this time the patient rather suddenly noticed a pounding noise in

the right ear while at church. She did not know to what the noise was due, but she found that she was able to control it by pressure over the upper part of the right side of the neck. This abnormal sound has been more or less constant since and is described as a surging in the right ear. The sound varies considerably in nature and is described as a swishing or jetting sound, or at times when less marked it may be noticed as a low-pitched murmur on the inside of the head. Lately, when severe, this surging or swishing sound seems to radiate upward and extend over the vault into the left side. On these occasions the sensation is like swirling water. This sound is rhythmic in character and is synchronous with the heart beat. It is aggravated by any sudden movement or on throwing the head backward, or turning it toward the left side. There has been a definite impairment of hearing in the right ear for some years according to the patient, but the present difficulty seems to have aggravated the condition.

The patient is a large, fairly well developed woman past middle age who does not present any unusual manifestations of ill health nor does she assume any position which is suggestive of any particular neurologic disease. Her illness has not incapacitated her in the carrying on of her household duties but she is very much concerned about the pain in her nose and face and the unpleasant throbbing sensations in her head, which more recently are associated with a similar sensation in the scalp at the top of her head. This rhythmic sound at times interferes with her sleep so that it is necessary to resort to sedatives.

*Neurologic Examination.*—There is no dilatation of the superficial vessels of the scalp. There is unusual tenderness of the scalp experienced to such a degree that patient has found it necessary to discontinue the use of hairpins. The percussion note over the skull is not particularly high pitched and examination of the cranial vault with a stethoscope discloses a characteristic murmur only intermittently. This murmur is audible to the examiner when a stethoscope is placed beneath the eye just adjacent to the nose on the left side. The cranial nerves were normal with the exception of the following.

*Optic Nerve.*—Patient has had a blurring of vision for the past two or three years which is indefinite in character. On one occasion recently, a band scotoma was apparently experienced by the patient as she stated that on looking at an individual's face, the lower part of the face and neck were not visible to her. Visual acuity was somewhat diminished and perimetric examination showed a moderate concentric contraction of the fields. Examination of the fundi revealed the veins to be somewhat overfull, particularly in the left eye, with early tortuosity. No elevation of the discs was noted.

*Trigeminal Nerve.*—There is a definite history of pain in the left side of the face over the eye or at the emergence of the infra-orbital division of the left trigeminal nerve, associated at times with a creepy feeling over the left side of the face. This pain is referred to the left upper lip and toward the left ear, and also in the left parietal region of the skull and the left palate. There are no objective sensory or motor changes in the face or involving the muscles of mastication.

*Acoustic Nerve.*—The patient has had a definite tinnitus in the right ear for nine months which is synchronous with the heart beat. Tests for auditory acuity with whispered and conversational voice with and without the Bárány apparatus and with the tuning forks revealed an approximate decrease in acuity of 20 percent in the left ear and 75 percent in the right ear, which was chiefly

of the conductive type, but definite damage in the higher registers was present. Tests of the vestibular apparatus showed normal reactions in all canals.

*X-ray and Laboratory Findings.*—Roentgenograms of the skull taken four months ago disclose a rather thin cranial vault with no evidence of increased intracranial pressure and no areas of erosion. The middle meningeal channels are rather large. There is one rather narrow pacchionian depression at the junction of the two coronal sutures. The sella turcica is slightly larger than average, is round in type and the anterior clinoid processes seem to be rather sharp. The pineal body, which is faintly shown on the lateral view, on actual measurement seems to be displaced somewhat forward.

Examination of the blood showed: Hemoglobin 100 percent, red blood cells 5,624,000, color index 0.89, leucocytes 7,900, polymorphonuclear neutrophils 54 percent (non-filament 3, filament forms 51), lymphocytes 38.5 percent, mononuclears 3 percent, eosinophiles 4.5 percent. The red blood cells were normal in size, regular in shape and took the stain evenly. No nucleated red blood cells were seen. Platelets were normal in number. Sedimentation time, (Linzenmeier tube) 12 mm., 50 minutes, 18 mm., 80 minutes. The Wassermann test was negative. The blood-pressure was taken on several occasions, the highest reading being 138/90.

#### DISCUSSION

There can be no question that the patient has a chronic suppurative disease in the accessory nasal sinuses on the left side which is the contributing cause of the pain and distress which she experiences. The reference of the pain and clinical findings would lead one to believe that the sphenoidal and postethmoidal sinuses are involved on that side producing some irritation of the sphenopalatine ganglion and giving rise to the characteristic symptom-complex.

The head noise of which she complains, we believe, is an entirely separate and distinct entity. In our opinion this is due to an aneurysm of one of the large vessels at the base of the brain on the right side. The findings are somewhat vague insofar as exact localization is concerned, but the limitation of the murmur in the right ear and the decreased auditory acuity on the right side point to its location as being in the right posterior fossa. In this case, the most probable site of origin is at the point of emergence of the posterior inferior cerebellar artery from the vertebral. An aneurysm in this region usually extends into the cerebellopontile angle on this side and gives rise to very definite auditory manifestations. This leads us to believe that an arteriovenous aneurysm of the cerebral substance can be definitely excluded.

There is very little to offer in the way of treatment of these intracranial lesions. We are of the opinion that ligation of the internal carotid artery would not prove of sufficient value to justify recourse to this procedure.



We have advised continued rest for a period of several months in the hope that the aneurysm may strengthen its walls sufficiently to prevent early rupture, inasmuch as the patient volunteers the information that any irritation or sudden movement accentuates the murmur.

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## DISCUSSION

CYRIL COURVILLE, M.D., Los Angeles, Calif. (by invitation): The subject of this paper is one of great interest to both otologists and neurologists. No doubt many instances of objective tinnitus or bruits escape us for want of auscultation of the head.

There are a number of intracranial vascular lesions which may be responsible for a bruit of sufficient intensity to be heard with a stethoscope applied to the cranial vault, to the globe of the eye or over the carotid arteries. Severe traumatism to the head occasionally results in rupture of the carotid artery into the cavernous sinus (pulsating exophthalmos) resulting in an arterio-venous aneurysm which causes an audible murmur. Congenital arterio-venous aneurysms of the brain are almost invariably accompanied by subjective and objective rhythmic bruits, synchronous with cardiac systole. Purely venous anomalies of the brain do not cause such sounds as a rule, although we have studied one case of a widespread dilation of the dural sinuses which provoked a very loud murmur heard over most of the cranial vault.

True aneurysms of the intracranial arteries as a rule are not accompanied by murmurs since most of them are small. Even in the larger saccular ones

no bruit may be provoked since the blood current is not sufficiently disturbed. When such an aneurysm occurs in the posterior fossa too, it almost invariably arises at the point of emergency of the posterior inferior cerebellar artery from the vertebral artery. In its expansion, it soon compresses the structures in the cerebellopontile angle including the nervus acusticus. A unilateral rhythmic murmur, audible both to patient and physician, is often provoked by such a lesion. The bruit is audible since it is in immediate proximity to the eighth nerve. This may be the situation in the case here considered.

In rare instances, highly vascular tumors of the intracranial structures such as gliomas, meningiomas or angiosarcomas may cause these adventitial sounds.

ANDREW A. LOVE, M.D., Los Angeles, Calif.: Tinnitus aurium has been discussed by many otologists since it was first recognized, and of all the symptoms which the aurist is called upon to relieve, it is the most elusive and difficult to control. The most useful classification, based on the underlying lesions or disorders is as follows:

1. *Obstruction sounds*, or noises due to occlusion or impaired mobility of some portion of the sound-conducting apparatus.
2. *Labyrinthine sounds*, or noises due either to structural changes in the cochlea, or to alterations—either increase or diminution—of intralabyrinthine pressure.
3. *Neurotic sounds*, or noises due to abnormal irritability of the auditory nerve.
4. *Cerebral sounds*, or noises due to abnormal conditions acting upon the auditory centers in the cerebral cortex. (Auditory hallucinations.)
5. *Blood sounds*, or noises produced by the blood current in vessels in or near the ear, and due to either disturbances in the local or general circulation or to abnormalities in the size, shape, or position of the vessels.

The first four types cause subjective tinnitus and the last one a tinnitus of both objective and subjective types.

An objective tinnitus, that is, one that can be heard by the examiner through the diagnostic tube, may be caused by two conditions. The first is a clicking or snapping sound caused by a rhythmic, spasmodic contraction of the muscles related to the eustachian tube which are the levator palati, tensor palati and tensor tympani. These sounds are not synchronous with the beat of the heart, however, and so are not of vascular origin, as the lesion in the present case undoubtedly is. The vast majority of cases of objective tinnitus are of vascular origin, and there are many pathological possibilities, of which the following is a fairly complete list. Valvular lesions in the heart itself, abnormalities of the carotid artery, the jugular bulb or of the blood vessels near the affected ear, may be the cause. In this case there is no valvular lesion of the heart. Abnormalities of the carotid artery occur but rarely but when present are centrally located near the pituitary body. If the objective tinnitus were caused by an abnormality of the jugular bulb or internal carotid artery, such as an unusually close proximity to the internal ear due to a dehiscence, the tinnitus would always have been present. In this patient the tinnitus is of but a few months' duration. Traumatic aneurysms of the temporal or occipital arteries may cause objective tinnitus which disappears on ligation of the affected vessel. There is no history of trauma in this case.

As is well known, the lateral sinuses on the two sides frequently vary in size, and it might be assumed that a large lateral sinus discharging into a small jugular bulb could set up an audible bruit, but in this event also the resulting tinnitus would have been present during at least the greater part of life. A brain tumor of vascular type must of course be considered, but as yet there are no definite signs to indicate this type of lesion.

The fact that rotation of the head will sometimes cause a cessation of the tinnitus and that sometimes it is necessary to turn the head from side to side to initiate it is an interesting feature of this case. It would seem that only slight changes in vascular pressure alter the mechanics of the situation.

This lesion is in all probability of unilateral occurrence because the tinnitus is only present in one ear. The fact that practically all other causes of objective and subjective tinnitus can be eliminated points to the probability that the cause is a cerebral aneurysm. The point of greatest possibility in these aneurysms is the juncture of the vertebral with the right cerebellar arteries.

ISAAC JONES, M.D., Los Angeles, Calif.: A strong man, age twenty-eight, was seized with a severe headache, entirely on the right side. I happened to be out of town and Doctor Lyster sent him to Dr. Gilbert Roy Owen for x-ray. Doctor Owen thought to cocainize his sphenopalatine ganglion. This gave immediate relief. The headache returned, and, a few days later, I also gave him relief by cocainizing the ganglion. I noted that he had a subsiding acute tonsillitis, and therefore told him not to worry—that everything would be all right—that he had involvement of the ganglion on the right side caused by the tonsillitis. He had so much pain, off and on, that he begged me to take his tonsils out at once. I told him that we must wait until all the tonsil inflammation had subsided. This was fortunate because, two days later, during sexual intercourse, he had a sudden agonizing headache and died in a few hours. The autopsy by Dr. George Maner showed a rupture of an aneurysm of the right middle cerebral artery, near the posterior communicating.

*Comment.*—My abysmal ignorance of the true diagnosis, the relief obtained by cocainizing the sphenopalatine ganglion, which admits of no definite explanation, and my surprise to have Doctor Maner tell us that such aneurysms are rather common, and his assurance that I was probably further in error, in that I thought this aneurysm was due to trauma from a severe blow on the head in an automobile accident ten months before his death—these are the important points in this case report.

Professor Knudsen and I studied a little girl who had such a loud *objective* tinnitus that one could hear it several inches away from her ear. We considered having this tinnitus recorded by a phonograph record in order that others who could not see the child might hear this tinnitus. We would have accomplished this, but at that time the technic of taking such records was not sufficiently advanced.

This tinnitus noise was due to the "kissing" of the moist surfaces of the mucosa within the right eustachian tube. At first, the little girl found that she could produce this noise in her ear voluntarily. This soon created a great interest among her friends and she naturally took pride in it and kept developing the procedure—until it went beyond her control.

During the months that we studied this child, this tinnitus was very annoying to her and she could not stop it at will.

## THE TREATMENT OF RHEUMATISM

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The gradually developing conception that rheumatism is a systemic disease due primarily to some error in our fundamental life that encourages infection has, it seems to me, tended to discourage the efforts of our surgeons to remove the foci of infection. This is so contrary to my feelings in the matter that I determined to review the subject from the standpoint of the work done in the otolaryngologic department of the Southern Pacific Hospital, where some 3,000 tonsillectomies have been done since 1927.

It is well known that rheumatism is a general term covering a multitude of pathologic lesions, each worker classifying the cases according to his best knowledge of the subject, but the classifications themselves give us a fine lead toward treatment and a hope for a better knowledge of the subject in the future.

Burbank and Hadjopoulos,<sup>1</sup> after a prolonged serologic study, made an illuminating classification of arthritis into:

(A) Arthritis reacting to hemolytic streptococci and belonging to the isotrophic class (periarticular).

(B) Arthritis reacting to hemolytic streptococci but of different fixing properties. Type anisotrophic or deformans.

(C) Arthritis reacting to streptococcus viridans type and belonging to the osteo-arthritic or productive form.

Although these soon became mixed types, these workers leaned strongly to the opinion that the disease was definitely of bacterial origin and specific in character; that is, that each organism produced primarily a definite type of lesion.

Hench, 1930,<sup>2</sup> classifies the disease into

1. Infectious.
2. Traumatic.
3. Senescent.
4. Chemical, *i.e.*, gouty.

This classification would seem to be ideal for treatment and seemingly would enlarge the field over that of Burbank.

Again, Cecil<sup>3</sup> calls attention to a more recent classification:

1. Infectious (rheumatic fever; rheumatoid arthritis; arthritis caused by specific organism).
2. Degenerative (osteo-arthritis or hypertrophic).



3. Allergic.
4. Traumatic.
5. Metabolic (gout, scurvy; rickets).
6. Neurogenic arthropathy (syringomyelia, Charcot's posthemiplegic, etc.).

The American Rheumatic Commission<sup>4</sup> has classified the disease into

1. Atrophic, including conditions known as proliferative, rheumatoid, ankylosing, metastatic and Still's disease.

2. Hypertrophic, including degenerative osteo-arthritis; menopausal, senescent, nonankylosing, von Bechterew syndrome and the Marie Stumpel syndrome.

They threw together the specific arthritides and those of gonorrhea, syphilis, gout, trauma, rheumatic fever, tuberculosis, tabes, hemophilia, purpura, etc.

The Ely and Fisher classification is :

		<i>Type I</i>	<i>Type II</i>
Goldthwait .....	X-ray appearance	Atrophic	Hypertrophic
Nichols and Richardson .....	Histogenetic Etiologic	Proliferative Bacterial Lues Tb. Neisser Staph. Strep. Etc.	Degenerative Protozoal (?)

Rheumatoid arthritis is Type I in histogenesis but no bacteriologic etiology known.

Marie Stumpel type is Type II in its histogenesis but has some of the late characteristics of Type I.

No subject can be approached with any great satisfaction that is accompanied by as great confusion in the nomenclature as is this, and still there is a glimmer of light through it all when one realizes that the individuals who have made the most intensive studies of the subject lean to the opinion that bacteria are at least a potent cause of the symptoms complained of and of the pathologic lesions present. That it may be a systemic disease is true, or that the general condition of the patient may determine the character of the local lesion or the rapidity of its attack, etc., but this is true of any other disease and not limited to rheumatism. The multiplicity of lesions found in tuber-

culosis and the varying intensity of attack is no more profound than is that of rheumatism, yet there is no question about the cause of the disease. The picture in the majority of the rheumatic cases is that of a bacterial invasion modified by the character of the host and the virility of the organism.

If there exists a constitutional derangement, such as lack of iodine, calcium, magnesium, sulphur or a too pronounced carbohydrate or protein diet or a dilated or elongated colon or a defective posture producing a trauma, as is claimed by many, it still remains that the ultimate lesion is caused by a bacterium and that this bacterium must be eliminated as soon as possible in order to give the host a chance to gain its equilibrium.

It is true that the body may become immune to the presence of bacteria, and if the body is provided with the necessary help the rheumatic tendency may be shunted, but there is no reason why that should be the main effort on the part of the physician in attendance if he once has a clear grasp of the ravages which may be meted out to the patient if the infection is not properly cared for. If we can show that there is a direct relation between a focus of infection and the diseased joint, in some cases it is more than proper to at least remove all foci as a preliminary to any other treatment, even though there is a doubt in the physician's mind as to the real origin of the disease.

To the 3,000 tonsillectomy patients at the Southern Pacific Hospital questionnaires were sent asking if the operation was done on account of rheumatism and, if so, what was the result. There were 1,257 replies (many of the men were no longer in our employ and did not get the paper). Of these, 819 were done for reasons other than for rheumatism. Of the remainder, 300 claimed they were entirely free of rheumatism, 100 were improved to various degrees and 38 claimed that there was no improvement.

Naturally the question arises as to how many of all these actually had what is called rheumatism; that cannot be ascertained, but it must be assumed that a large proportion knew what they were coming to the hospital for. Most of these cases were among people of from thirty to fifty years of age, males, and the majority from an intelligent class. On reviewing the histories of those claiming no improvement, I found that the larger proportion had had their trouble for many years and showed deformities by the x-rays. The histories of some of this group did not mention any rheumatic pains but stated they entered hospital for tonsillectomy on account of repeated sore throats. On questioning the various members of the staff, I found

it was a unanimous opinion that the removal of the foci had in the majority of cases been the prime factor in the cure of the cases.

My own observation, and I have questioned many minutely, is that there occurred immediately on removal of the tonsils an amelioration of pain or swelling or of both; this often occurred before the patient reached his bed, and that certainly is what one could expect if the pain is due to a toxin circulating in the blood stream specific for a certain joint. I have had acute cases come in on crutches and leave the operating room without them. Many of these cases did not get well until infected teeth were removed or the prostate gland was treated or a nasal sinus cleared out, but this is only confirmation of the opinion that the immediate cause of the joint lesion was an infection.

One of my cases was a laryngologist who was gradually becoming helpless. He had, over a period of years, hunted everywhere for a focus. He had never had sore throats and his tonsils were so small and innocent looking that both he and I doubted their being the origin of his trouble. At operation I opened an abscess as large as one's thumb nail and at once all arthritic symptoms disappeared so that within six months there was normal movement of the spine and limbs. This is not an isolated case, but well illustrates the point I wish to make, that there is some definite relationship between a focus of infection and a pathologic lesion in the joint.

I purposely made no attempt to separate the cases with the atrophic from the hypertrophic forms, preferring to let the patient himself say yes or no as to the presence of what is popularly known as rheumatism, thus getting a cross section of the results that might be obtained through removal of focal infections. This may not be scientific but it proved to be highly illuminating, for it must be granted that in the class of cases operated upon the disease that is most common is the osteo-arthritis and not rheumatoid arthritis, and the staff of the hospital agrees that the majority of the cases seen were of the former type.

Conceding that the rôle of a bacterium is important, at least in the production and, therefore, the treatment of arthritis, it is just as necessary to go further and inquire into what causes the infection to become manifest and into the possibility of controlling that factor.

On questioning physicians who have practiced in the Philippines and in Japan, I find that there is very little rheumatism in these places. It has been suggested that in neither place are proteins consumed in anywhere near as large quantities as in America or Europe and that this large protein consumption in the latter countries pro-

duces a fermentation in the bowel which in turn causes a swollen mucous membrane and glandular system with its subsequent train of infectious sequences. Keeping this in mind, one can well understand the desire of one group of men to cure by removing the colon or by high enemas.

Another suggestion has been made that it is the dominance of iodine in the Eastern diet that decreases the incidence of rheumatism. Again, in these countries, there was formerly a preponderance of foods untampered with by manufacturing processes: whole rice, mushrooms, greens, fish, fruits, instead of prepared grains from which all the important and necessary qualities have been removed, canned milk, vegetables, preserves, sugared goods, frozen meats, that have hung for a month, etc.

When we think of what happens to a kernel of wheat as it is prepared for white flour we wonder that there is any nutrient value left. What we are handed out is pure starch bleached by  $H_2SO_4$  in order that it will remain dry for shipment. Vitamin, oil, roughage, etc., have all been removed. If we could grind the raw kernel as in former times a great many of our modern diseases might take on a different appearance, and this applies to a large list of prepared foods.

The result of feeding prepared foods to chickens is well illustrated by the experiments carried out by one of the patrons of Park and Pollard, "who divided a hatch of 1,000 chickens, placing 500 in one brooder and the remaining 500 in another brooder in adjoining pens, under as identical conditions as possible, with the exception of the feed, the first pen being a commercial ration overmineralized with inorganic materials. We say 'overmineralized' for the reason that a later test of the feed developed a 12 percent ash content in the ration. This group of chickens was very nervous, lacked one or two points of pigmentation in the feet and beaks, as compared with the second pen, was as flighty as young quail, fearful of shadows passing across the pen and could not be caught except by panicking them into a corner and then seizing a handful of them. The second group, fed upon a simple grain ration containing liberal quantities of cornmeal and our seaweed product, was more tame than the customary group of Leghorn chickens, in fact, at any time the attendant—a woman—bent over in the pen, the youngsters were gathered around and picking at her hands and talking to her as contented chickens always do. The pigmentation in the beak and legs was as previously noted, two shades or more deeper than the other lot. They carried their heads lower naturally, and were very easily handled



and broke to roost very easily, whereas the other pen would not content themselves on roosts until two or three weeks later."

If this occurs in chickens, the same or similar reactions undoubtedly occur in the human, and the fine studies now being made on the various constituents of our food products, such as the iodine in the milk, the mineral content in vegetables from different parts of the country and numerous other investigations into the food intake are going to have a profound influence in our conceptions concerning rheumatism.

It is still possible that, as in beriberi, we may turn to the East for a solution of the problem as to how to attack the fundamental cause of our infections, but until that time we shall have our enthusiasts using calcium ortho-iodoxy-benzoate, colloidal sulphur, gold, HCL, bicarbonate of soda, salicylic acid, vaccines, vitamins, internal gland secretions, soluble antigen and x-ray, but in my experience the thing that will save the greatest time for the present still remains the thorough elimination of the foci of infection.

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## TONSILLECTOMY IN THE TUBERCULOUS PATIENT

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An extensive experience with tuberculous patients as attending otolaryngologist to a large institution caring for such patients has convinced the writer of the importance of tuberculosis of the tonsil as a complication of pulmonary tuberculosis. There seemed to be no good reason why these depots of tuberculous disease should not be actively treated in the manner that other similar complications are, that is, by eradication. The general medical staff was consulted and felt that no objection of any logical validity existed to tonsillectomy in properly selected and adequately controlled cases. For the past five years, therefore, the writer has done tonsillectomies on tuberculous patients and offers what follows as a survey and summary of this work. The work done during the year 1930 has been excluded from this report because no histological sections were made of the excised tonsils. The material on which this report is based comprises 102 tonsillectomies done on adults under local anesthesia and forty done on children under general anesthesia. It was thought that a presentation of the facts brought out by a survey of our cases would be of value to both the clinical and the pathological view of the disease.

The writer is well aware of the recent appearance of a most excellent paper on this subject by Newhart, Cohen, and Van Winkle, has read this paper carefully, and makes no pretense to a more comprehensive treatment of the subject than can there be found. The material here presented, however, has been accumulated contemporaneously with that of the authors mentioned and the results agree so closely with theirs that, even if they only serve to emphasize points already well made, the author will be satisfied.

An excellent historical summary is contained in the paper already mentioned and will not be repeated here. The subject has been explored from two standpoints: the incidence of tuberculosis of the tonsil in non-tuberculous individuals, and that in the tuberculous ones. The former has been summarized by Newhart, Cohen, and Van Winkle on the basis of 30,678 cases reported in the literature. In this large group, 625 cases of tuberculosis of the tonsil were found, or 2.03 percent. The individual reports gave figures that varied

from 1 percent to 5 percent. In tuberculous individuals, the figures are quite different. Here the figures vary from 20 percent to 90 percent and depend upon the stage of the disease and the infectivity of the sputum, to mention only the two most important and positive factors. In Newhart's series of 112 cases, 42 percent had positive tonsils.

From the foregoing, as well as the reports of such writers as Boone, Callaway, MacReady and Crowe, Mullin, Newhart, and Wood, it is abundantly clear that tuberculosis of the tonsil is found with startling frequency in both adult and juvenile pulmonary tuberculosis and forms an unnecessary obstacle to the complete recovery of the tuberculous individual. Add to this the non-tuberculous complications in which the tonsils may be implicated, ranging all the way from the functional psychoses to peritonitis (F. Riedel, cited by Vajda) and the logic of the stand for their removal in selected cases becomes overwhelming.

A word on our method of selection and preparation of patients seems in order. We feel strongly that the whole procedure stands or falls on the care with which this and the subsequent operation is done. The selection of cases was always made in collaboration with the staff at the Leahi Home. The patient was seen primarily in the Ear, Nose, and Throat Clinic and, if the tonsils were infected, markedly enlarged, or the seat of an ulcerative tuberculous process, or for any other reason considered a hazard to the patient from the special ear, nose, and throat view, he was referred to the medical department for consultation. Here the chief points considered were the extent of tuberculous involvement; its general trend toward healing or progression; the activity of the lesion as evidenced in recent x-ray, sputum, and blood studies; and the type and extent of tuberculous complications. If a careful weighing of these factors against the tonsil disease indicated that tonsillectomy could be done without affecting the patient's condition unfavorably and with the possibility of removing an obstacle in the way of his recovery, this was done at the earliest opportunity and in the manner briefly outlined below.

We select our surgical cases from those patients whose progress toward recovery, we feel, is hindered by pathology of the tonsils, and we agree with Newhart, Cohen, and Van Winkle that a tuberculous patient should not be denied the benefits of tonsillectomy simply because he has tuberculosis.

A few authors have discussed the technic of tonsillectomy in tuberculous individuals (see Boone, Hollender, Levy, Newhart, Cohen, and Van Winkle). We have seen no indication for electro-

cautery and have adhered in detail to the following routine in all local tonsillectomies.

(1) Only those patients are operated upon whose tonsils demand enucleation because of definite gross pathologic changes, or whose history indicates recurring acute attacks of tonsillitis. No patient has been operated upon until he has been carefully examined by the general staff and considered by them to be a good risk. No hopelessly advanced patient is considered.

(2) Study of the psychological state of the patient is carried out. The patient is not rushed into operation, but every effort is made to gain his confidence, so that in the end patients do not fear the ordeal. We have observed that tuberculous patients usually gag less than non-tuberculous patients, and we attribute this to the fact that these patients have been taught how to cooperate with the examiners by the many pharyngeal and laryngeal examinations that always precede the operation. Heavy hands and awkward manipulation will cause any patient to gag.

(3) Clotting and bleeding time is carefully checked up and calcium is given when indicated. The operation is delayed until the bleeding and clotting time is brought within normal limits. In forty-three cases which served as a basis for a statistical analysis of clotting time, this varied between three and eleven minutes and averaged five and three-tenths minutes. The individual with a clotting time of eleven minutes had moderate bleeding from both tonsil fossæ for about three days following his operation.

(4) Routinely, unless definitely contraindicated, it is our practice to give our patients one hour before operation one or two capsules of sodium amytal, depending upon weight and age.

(5) We never spray the pharynx with cocaine, as we rarely find it necessary, and we believe that the use of such toxic medications should be reduced to a minimum. If there is reason to believe that the patient will gag too much in the preliminary infiltration stage, the patient is allowed to suck on a tablet of euphagin fifteen minutes before starting infiltration. The pharynx is painted with a solution of 5 percent mercurochrome in acetone-alcohol. Five c.c. of 1 percent novocain is infiltrated into each peritonsillar region in the appropriate places. It is exceptional, indeed, if more than 5 c.c. is necessary to insure an absolutely painless operation.

(6) The patient is always seated erect and before the operator in a nose and throat treatment chair with arms. The patient holds his own sputum basin, which gives him something to do with his hands, and aids in his cooperative attitude. The head is tilted forward



and downward to allow mucus, blood, and débris to escape after the removal of each tonsil.

(7) Blunt and scissors dissection is routinely used, a number nine wire is used and the snare is slowly closed in order to crush the bleeding points, thus reducing the bleeding to a minimum. If excessive bleeding occurs, pressure is not used, but the bleeding point is immediately grasped and crushed. If this fails, 000 catgut on a fine curved non-cutting needle is passed under the bleeding point and tied.

(8) The patient is returned to his bed by litter or wheel chair, and a hypodermic of morphin  $\frac{1}{8}$  to  $\frac{1}{6}$  grain with atropin  $\frac{1}{200}$  to  $\frac{1}{180}$  grain is given. No diet is given for fifteen hours, but ice chips may be given if the patient desires. Aspirin gargles are given fifteen minutes prior to eating and the patient is immediately started out on a good nutritious soft diet. The tonsillar fossæ are painted once daily with tincture benzoin compound with one grain of mercury bichlorid to the ounce. This antiseptic aids in sealing the raw open surface and encourages the deposit of fibrin in the fossæ. Every effort is made to keep up the body weight, so often lost after tonsil operation, and the conservation of which is so vital to a tuberculous patient, by rest in bed and nutritious diet.

In reviewing our statistics it was thought best to exclude the local tonsillectomies done on employees and other non-patients and confine them to the sixty-three adult patients, among whom there were nineteen tuberculous tonsils, or 30 percent, and the forty children, who yielded one positive case, or 2.5 percent.

TABLE 1.—SIXTY-THREE TONSILLECTOMIES, 1931-1934

1931 .....	25
1932 .....	15
1933 .....	15
1934 .....	8
<hr/>	
Total .....	63

TABLE 2.—AGES: TWELVE YEARS TO FORTY-THREE YEARS. AVERAGE AGE: TWENTY-FIVE YEARS

Sex:	
Females .....	29
Males .....	34
<hr/>	
Total .....	63

TABLE 3

<i>Nationality*</i>	<i>Number</i>	<i>Positive</i>	<i>Tonsils</i>
Japanese .....	31	13	42%
Chinese .....	6	1	16%
Filipino .....	6	1	16%
Hawaiian and part Hawaiian...	10	3	30%
Portuguese .....	5	..	....
Caucasian .....	3	1	33%
Porto Rican .....	1	..	....
Korean .....	1	..	....
Total .....	63	19	30%

If only those patients who had some form of pulmonary tuberculosis are considered, these nineteen positive were obtained from fifty-four cases or 35 percent. The group with pulmonary tuberculosis was almost evenly divided among minimal, moderately advanced, and far advanced, the numbers being 18, 18, and 16 respectively. From the eighteen minimal cases, four positive tonsil sections were obtained, or 22 percent; from the eighteen moderately advanced patients, seven, or 39 percent; and from the sixteen far advanced, eight, or 50 percent. This agrees very closely with the figures of Newhart, Cohen, and Van Winkle, which are 17, 44, and 57 percent.

TABLE 4

<i>Status</i>	<i>Number</i>	<i>Positive sputum</i>	<i>Positive tonsils</i>
Minimal .....	18	5	4
Moderately advanced .....	18	8	7
Far advanced .....	16	13	8
Arrested inactive .....	3	..	..
Bronchiectasis .....	1	..	..
Non-spec. pneumonia .....	2	..	..
T. B. keratitis .....	1	..	..
Lung abscess .....	1	..	..
Chronic tonsillitis .....	2	..	..
Tuberculous hip .....	1	..	..
Total .....	63	26	19

When a correlation is made between tuberculous tonsils and sputum positive patients, it is found that twenty-nine of these patients supplied fourteen positive tonsil specimens, whereas, in thirty-four

sputum negative (negative) patients the tonsils were positive only five times. Forty-eight percent of the sputum positive cases, therefore, had tonsil tuberculosis as compared to 15 percent of all others. The significance of this relationship will become more apparent in connection with discussion of the pathogenesis of tonsil tuberculosis.

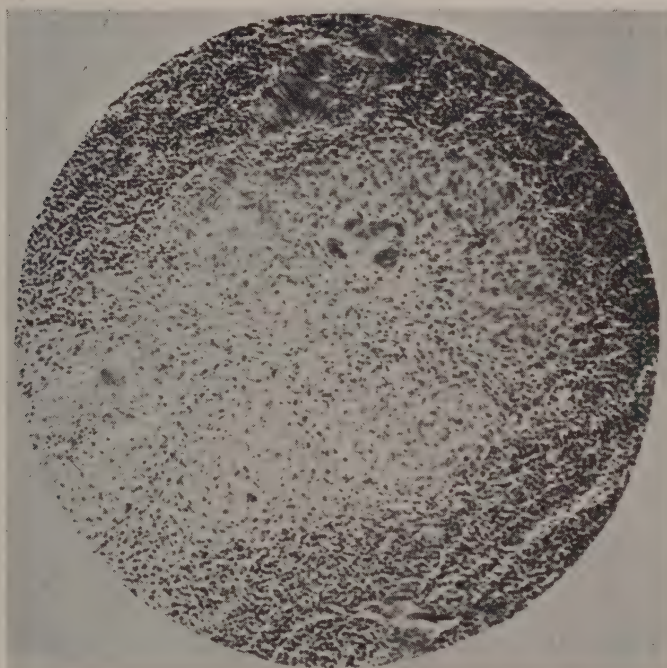


FIG. 1. Deep tubercles with giant cell formation.

Of the four individuals with tuberculous tonsils in the minimal group, two had positive sputum, or 50 percent. Of the seven in the moderately advanced group, five had positive sputum, or 70 percent; and of the eight in the far advanced class, seven had positive sputum, or 87 percent.

TABLE 5.—TUBERCULOUS TONSILS AND SPUTUM

<i>Status</i>		<i>Negative sputum</i>
Far advanced .....	8	1
Moderately advanced .....	7	2
Minimal .....	4	2
Total .....	19	5

This emphasizes the correlation between tuberculosis of the tonsil and both extent of pulmonary involvement and bacilliferous sputum. For the rest, the tables are more or less self-explanatory. There was no case of tuberculosis of the tonsil found in a non-tuberculous individual

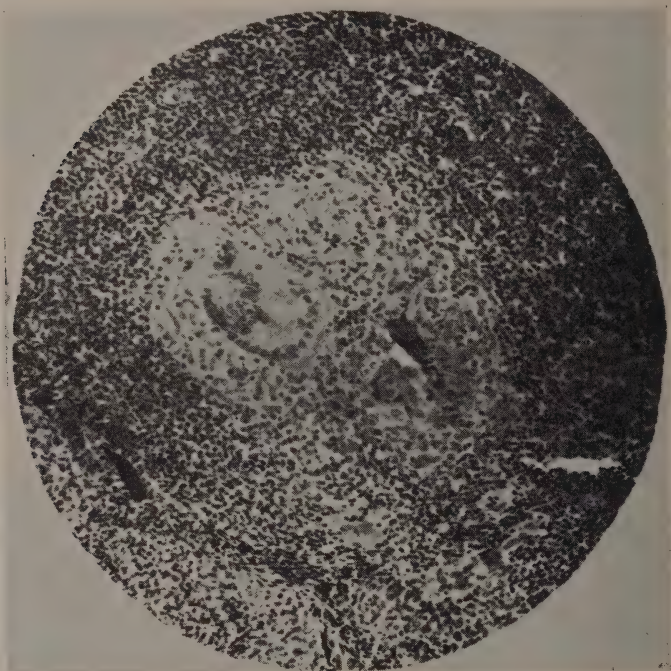


FIG. 2. Deep tubercle with giant cell formation.

In discussing the pathogenesis and pathology of tonsil tuberculosis, the question of primary tuberculosis of the tonsil may be ignored because our concern here is solely with tonsil tuberculosis as a complication of pulmonary tuberculosis. It is the prevailing opinion among most modern writers that the most important factor in the pathogenesis of tuberculosis of the tonsil is the presence of bacillus laden sputum. This sputum invades the crypts of the tonsils, which catch and may hold it for a considerable time. According to the experiments of Murata and Goerke (cited by Vajda), invasion of the tonsil proper cannot take place through an intact epithelium. Theoretically, therefore, a previous infection or injury of the cryptal epithelium is a prerequisite of tonsil tuberculosis. That this condition is easily met in the vast majority of adults can hardly be doubted. Cryptal involvement is considered by most authors (Weller, Mullin,



Fischer, Heubschman, Murata, Newhart) to be the commonest type of tonsil tuberculosis; C. V. Weller classifies the pathological changes found into three types: (1) Crypt infection, (2) ulcerative lupus-like lesions, and (3) diffuse miliary tuberculosis. According to Vajda, many authors believe that tuberculosis occurs only in hyperplastic tonsils. He cites the experiments of Mitschell and Dienlafory to prove this, the latter of whom obtained eight positive results among sixty-one cases using the method of injecting hyperplastic tonsil tissue extracts into guinea pigs. In our series there seemed to be a preponderance of this deep hyperplastic type (see Figures 1 and

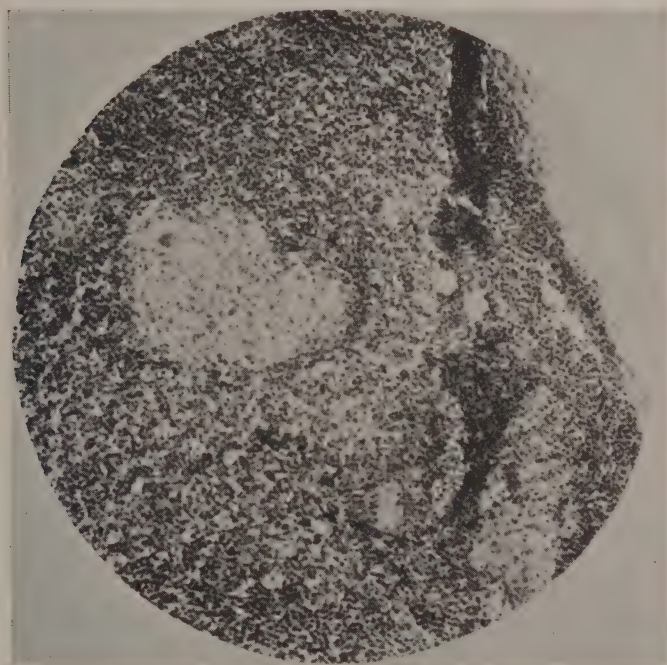


FIG. 3. Cryptal tuberculosis.

2 and 7) of tuberculosis over the superficial cryptal variety (see Figures 3 and 4). The miliary type was present in only one case. The others fell into either the cryptal or the deep hyperplastic type. We also had two cases of the caseo-ulcerous type (see Figure 5) in each of which the ulcerative process had destroyed practically all of one tonsil and part of another. In one of these cases there was extensive invasion of the faucial pillars and pharyngeal wall by the ulcerative lesion, illustrating well the correctness of the term "lupus-like" for this process.

We did not content ourselves, as did Newhart, with histologic sections alone, but did smears, cultures, and guinea-pig inoculations on a great many of our tonsil specimens. A total of 119 histological sections, eighty-five cultures and nineteen guinea-pig inoculations were made. We believe that serial sections of the tonsils would undoubtedly have increased our percentage of positives, but such a tremendous number of sections would have been too great an undertaking for our purpose. Three of our positive tonsil specimens showed positive cultures, one was positive on smear, and one positive on guinea-pig inoculation. The difficulty of the technic of



FIG. 4. Small tubercle subcryptal.

making cultures and guinea-pig inoculations is chiefly responsible for the comparatively poor corroboration offered by these methods. Caseation, which is held to be rather uncommon in tonsil tuberculosis, was found in six of our nineteen cases, or about 32 percent.

A very interesting case that deserves special mention was one of positive tuberculosis of the tonsil in a male child of nine who was admitted with a bilateral phlyctenular keratitis. His chest x-ray showed a left sided apical tuberculous process of the childhood type. After the eye condition had quieted down, tonsillectomy was done

and both tonsils were found riddled with small tubercles of miliary type (see Figure 6). This case can be explained best on the basis of a hematogenous dissemination, since all of the sputums were negative. It is further remarkable because of the fact that the literature available to us contained only one similar case, described by C. M. Jack, in a girl of eighteen. He also says, "No similar case could be found reported in the literature."

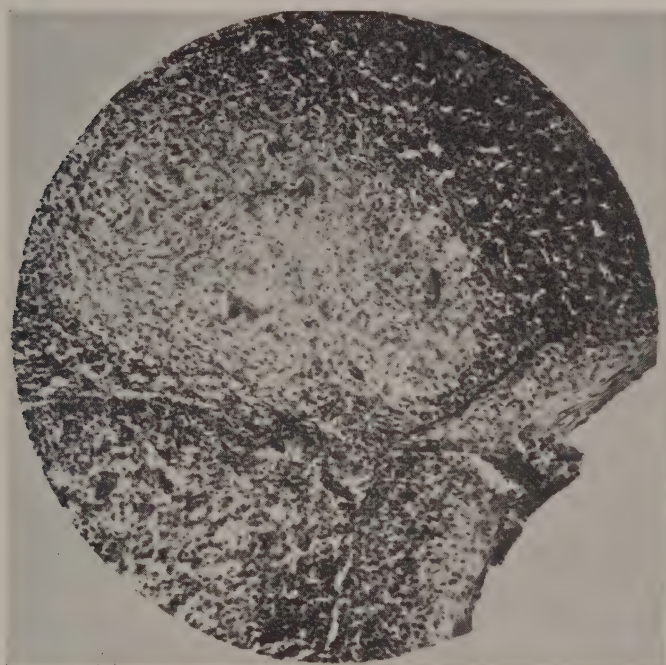


FIG. 5. Partly caseated tubercle just beneath epithelium of tonsil.

A correlation between the positive tonsil cases and their lung lesions revealed the following:

TABLE 6

	<i>Tuberculous lung lesion</i>	<i>Tuberculous tonsil lesion</i>
Bilateral .....	7	8
Right .....	8	6
Left .....	3	5
Indefinite .....	1	..



The lung lesion was ulcerocavernous in six cases, productive in eight cases, and in five, effusions with little parenchymatous involvement were present. The tonsil and lung of the same side were involved five times, three on the right, two on the left.

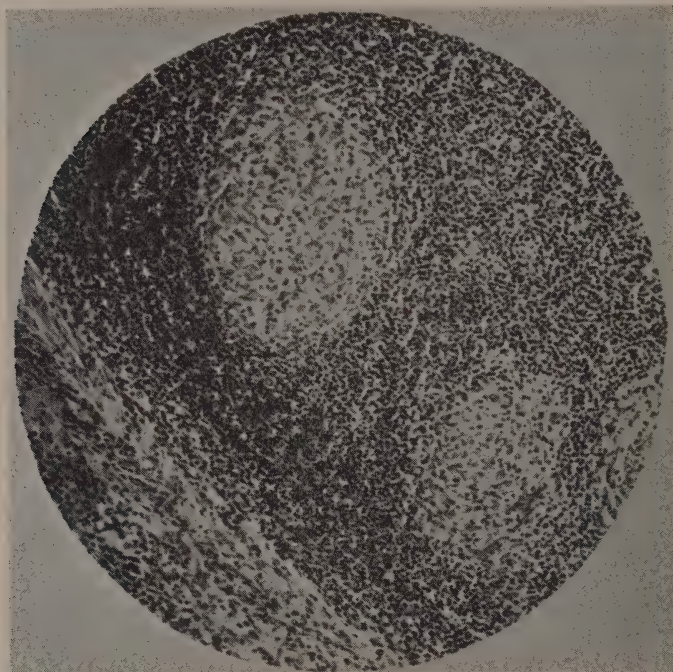


FIG. 6. Miliary tubercles deep in tonsil.

TABLE 7.—SIXTY-THREE TONSILLECTOMIES, 1931-1934

<i>Results</i>	<i>Tuberculous</i>	<i>Non-tuberculous</i>
Quiescent .....	27	..
Apparently arrested .....	13	1
Arrested .....	4	..
Improved .....	2	5
Unimproved .....	1	..
Died .....	2	..
In hospital .....	6	..
Cured .....	..	2
Total .....	55	8 = 63

The results of our work over a period ranging from eight months to nearly five years are apparent from the tables. Only the two deaths need clarification. One of these occurred three years after



tonsillectomy in a patient who died as a result of an anterior thoracoplasty. Though she was classified as far advanced and "sputum positive," her tonsils were negative (mistaken diagnosis). At autopsy she proved to have a dermoid cyst of the lung with no evidence of tuberculosis. The other occurred in a young girl seventy-five days after tonsillectomy, death being due to heart failure. The sputum of this patient was negative in the hospital.

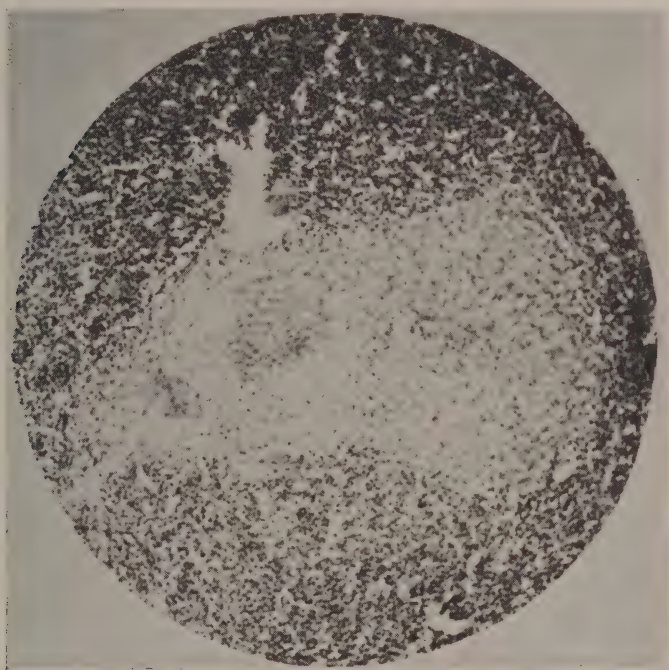


FIG. 7. Early epithelial cell tubercle.

### CONCLUSION

It can be concluded from this study that:

1. Tonsillectomy in properly selected tuberculous individuals is of considerable benefit in speeding convalescence.
2. No inherent danger lies in the operation when it is performed carefully, expeditiously, and by a skilled operator.
3. Tonsil tuberculosis as a complication of pulmonary tuberculosis is quite common and its frequency directly proportional to the extent of disease and the presence of bacilliferous sputum.
4. The frequency of tonsil tuberculosis and its significance as a concomitant of lung tuberculosis makes it highly important that

all adult cases on whom tonsillectomy is contemplated be explored by a competent internist for possible lung involvement.

*Note.*—The writer acknowledges with thanks and appreciation the valuable assistance of the medical staff of Leahi Home. Without such cooperation this five-year summary would have been impossible.

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## THE OPERATIVE TREATMENT OF NASAL SINUS DISEASE

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A discussion of the operative treatment of nasal sinus disease necessitates the correlating of various technical endeavors, some as yet unpublished, and the evaluating of methods and hoped-for improvements with which I have been occupied since the publication of my original article entitled "Fronto-ethmo-sphenoid Operation Under Local Anesthesia," in the *Archives of Otolaryngology*, in November, 1926. I am inclined to quote from that article as follows, because what was said then needs even more emphasis today:

"Indications for operation. The operative treatment of sinus disease is a subject that I feel should be approached with considerable caution. I am proposing a rather radical line of procedure that I feel may add to our efficiency in handling certain cases. Still I do not wish to leave the impression that I am applying these measures except after careful study or when former unsuccessful operative work would seem to make their applicability advisable. I have gone into the question of diagnosis frankly, but must make the point plain that the diagnosis arrived at does not condemn the patient to operative measures. It becomes a matter of the nicest judgment on the part of a well-trained surgeon, probably with the invaluable assistance of the internist, to determine whether the sinuses should be operated upon. A rubric of the operative indications would be only about as useful as those that have been compiled to tell us when to operate upon a chronic running ear. Many people with apparently excellent health are sufferers from infected sinuses. The hawking and spitting that is so commonly heard, and the mucopurulent expectoration seen, are evidence of the widespread distribution of sinus disease. While admitting that many people seemingly well are carriers of this infection, it is obvious that they are apparently in good health in spite of the sinusitis. It is a menace constantly carried about, and often proves to be the vulnerable heel of Achilles, leading to the establishment of some important vital disorder. The inability to discover the true focus, the removal of the wrong focus or the incomplete treatment of the right focus, have brought a certain opprobrium on the focal infection theory. Incomplete treatment of the right focus is practiced so commonly in connection with sinus disease that the specialists must take the onus of the lack of understanding on the part of many of the medical profession of the importance of sinus infection. These men have conscientiously and repeatedly referred patients for the elimination of nasal infection. Failure even after nasal sinus operation to cure the disorder in which the internist was interested, very frequently has been interpreted as indicating improper diagnosis. The use of the method I have advocated of gathering the nasal and postnasal discharge from the patient on pieces of cloth will show the internist himself whether the results he has asked for have been achieved.

"Ordinarily, before operation on the sinuses is to be considered, all other factors that may be contributing to the trouble should be corrected; climate has been given especial prominence. Whatever therapeutic measures have been found useful may be employed.

"Constructive surgery, when indicated, including submucous resection, tonsillectomy, and adenoidectomy should be given precedence and followed by sufficient time for accomplishment of results before the destructive surgery of the sinuses is advocated."

In presenting my present operative plan, it may be said that the intricate technical developments have followed naturally upon the introduction of a bloodless technic. This was accomplished by ligation of the vessels supplying the operative field, under local anesthesia secured largely by blocking the nerves before or at their entrance to the field. Sinus surgery may well be divided into a pre- and a post-ligation period. The literature of the former contains many descriptions of the bloody operative field where all nicety of technic was impossible. General anesthesia was then, and often still is, used routinely in the performance of sinus surgery, via external approach.

#### TECHNIC OF THE FRONTO-ETHMO-SPHENOID OPERATION, UNDER LOCAL AND BLOCK ANESTHESIA

Preparation of the patient:

*Analgesia.*—Sodium amytal is given by mouth; scopolamin and morphin hypodermically. Scopolamin gr.  $\frac{1}{150}$  to  $\frac{1}{100}$ , in my experience, has not caused the nervous reaction described by some.

*Anesthesia.*—Cocain is used locally in the nose by topical application. A fine wire applicator wound with a small bit of cotton is moistened with adrenalin and dipped lightly into flake cocain. Only what dissolves is taken. This gives a strong fresh solution, not cocain "mud." The nose is never packed with cocain saturated material because the danger of absorption is a real one. Novocain is injected superficially and deep in the skin. A long needle is then passed into the orbit to block the branches of the fifth nerve, the supraorbital, supratrochlear and the ethmoidals.

*The Skin Incision.*—The skin incision follows the curve at the inner angle of the orbit, extends below the unshaved eyebrow outward not farther than the supraorbital notch—artery thus avoided—and downward onto the nasal process of the maxillary bone. A length of 3 or 4 cm. ordinarily gives enough room because of the flexibility of the soft parts. However, as the scar becomes practically invisible, as much room may be taken downward as required. Ordinarily the frontal sinus can be cared for through this incision. There



are plenty of cases, however, where the frontal sinus is so large—extending to the external angular process of the frontal bone—that it cannot be adequately handled through this cut, nor will such a sinus drain properly into the nose as is expected according to the classical technic. In such cases, I use a supplementary incision at the outer angle of the orbit underneath the eyebrow, 3 to 4 cm. long as required. This incision permits attention to the lateral part of a large frontal sinus and will be discussed later.

*Opening Through the Bone.*—The periosteum is elevated, exposing the antero-inferior wall of the frontal sinus, and the frontal process of the maxillary bone. The lacrimal sac is partly raised from its fossa and the periosteum from the anterior few millimeters of the lamina papyraceæ. The anterior ethmoid artery and nerve are perhaps seen in the depth as the periosteum is raised from the lamina.

The lower end of the bony window is cut through with a sharp chisel. This cut is made first while the bone is attached and firm as possible. Care is taken not to cut or injure the underlying nasal mucosa which is to be preserved to form a frontal flap. The upper end of the window is made by entering the frontal sinus at the inner angle of the orbit. The medial limit of the window is carefully determined by study through the frontal sinus opening and through the nose. This bone incision should be as central as possible and still avoid thick septal and frontal bone. It is made with a sharp, flat chisel.

The fourth side or lateral wall of the bone fractures easily at the thin laminal edge. The bone as it is removed is carefully separated from the valuable nasal mucosa. The inner surface of this square of bone is pitted by part of the anterior ethmoid cells. The bone is thus quickly removed and admittance gained to the frontal, ethmoid and sphenoid sinuses.

*Control of Bleeding.*—It has long been recognized in general surgery that adequate control of bleeding is essential to proper surgical work. It was apparent that the application of this fundamental surgical principle was imperative if nasal surgery was to progress. In 1920, I managed to ligate all of the vessels of consequence that supplied the nose and accessory sinuses. These ligations, limited exclusively to the vessel supplying the operative field, are accomplished through the operative incision and in the immediate neighborhood of the surgical field.

Prior to these ligations the rhinological surgeon depended upon the local application of cocaine and adrenalin for ischemia. The complicated and difficult technic herein described can only be carried out

under the complete control of bleeding. The vessels that require ligation in the work under discussion, the fronto-ethmo-sphenoid operation, are the ethmoid arteries in all cases and the sphenopalatine artery when it is necessary to remove the floor of the sphenoid sinus. I have recently used the high-frequency coagulating current in handling these vessels. This may be found to simplify the technic materially if reliance can be placed on such control of these rather large vessels.

The usefulness of ligation of the ethmoid vessels, where they are at fault, in severe, otherwise uncontrollable nasal hemorrhage, is advocated. The usual last minute stampede to control the nasal bleeding from this source by ligation of the external carotid artery is misdirected. These vessels are branches of the internal carotid artery via the ophthalmic artery.

The ethmoid arteries differ anomalously in size, number and location. They are ligated, as originally described, by separating the orbital periosteum from the frontal bone and the lamina papyracea, and passing the ligature around the vessel as it passes medially through its foramen covered by orbital periosteum. They are then cut on the nasal side of the ligatures.

*Treatment of the Frontal Sinus.*—The frontal sinus has been opened in forming the bony window as described. Even if the orbital sinus is not diseased the removal of much of its floor is necessitated because the ethmoid cells so often extend orbitally beneath it. The frontal sinus is, therefore, always included in this operation and the provision for its drainage must be provided. This responsibility gives pause to the thoughtful surgeon to weigh carefully all considerations before operating.

The entire floor of the front sinus is removed. It forms much of the orbital roof. It is impossible to accomplish this through the original incision without undue trauma to orbital contents when the frontal sinus extends laterally to an extreme degree. Therefore, in such cases, a counter incision as already described, is made external to the eyeball. This supplements the original incision to permit the complete removal of all of the floor of an extremely large sinus. It serves the added purpose of affording dependent drainage for the outer part of a very large sinus which could never drain successfully over the curve of the orbital contents and down into the nose, as required by the single incision commonly employed. Temporary drainage in this locality gives results otherwise unattainable and the scarring is negligible.

When anatomic anomaly in the shape of a deep pocket in the extreme lateral position precludes the possibility of a cure of this

part—and consequently all—of the frontal sinus by drainage alone, the outer part of the orbital ridge is removed through the external or supplementary incision. The external part of the frontal sinus is thus obliterated. This does not cause much disfiguration. It is a thoroughly practical and valuable modification.

The lining of the frontal sinus should be removed as completely as possible if diseased.

*Treatment of the Ethmoid Sinuses.*—The ethmoid cells are removed carefully under direct vision. Those extending laterally are reached and obliterated by removal of the orbital roof where it forms the floor of the cells. These cells are removed cleanly up to the roof or dural plate and back to the sphenoid. The mucosa of the nose and that covering the medial wall of the ethmoid mass is carefully guarded while the underlying bone, including the superior and middle turbinates, is removed. This mucosa is used in forming flaps, as will be described.

*Treatment of the Sphenoid Sinus.*—The sphenoid sinus is entered through its thin anterior wall. The condition of the lining mucosa is carefully studied. If the mucosa appears normal it is not disturbed. The anterior wall or sphenoturbinates is removed, care being taken not to injure the sphenopalatine artery.

If, on inspection, the sphenoid mucosa is obviously diseased or the clinical symptoms demand its removal, a difficult problem presents itself.

The literature of the pre-ligation period is filled with evidence of the trouble that was encountered in the surgical treatment of the sphenoid sinus when the lining was diseased. Much stress was laid upon the difficulty encountered when that lining was curetted or removed. Granulation tissue, polypoid, purulent masses defeated every effort. We read warnings against tampering with the diseased mucosa because of the repeated occurrence of purulent polyposis or the formation of purulent crusts. It is written that crusts are the necessary consequence of too much nasal space.

In refutation of this fallacy the lesson might have been learned from common experience in general surgery. An incompletely drained abscess elsewhere in the body gives rise frequently to a fistulous tract lined by exuberant, polypoid granulation tissue. Neither polyposis nor crusting occurs in a nose where drainage is correctly established.

The sphenoid sinus is often a large obliquely placed cavity. Removal of the thin anterior wall or sphenoturbinates did not suffice to afford drainage. Attempts to remove the floor necessarily entailed

damage to the sphenopalatine artery. Hemorrhage from that large artery stopped further operative nicety. Further progress in the treatment of the sphenoid sinus demanded, first, control of the sphenopalatine artery.

The method developed for the ligation of the sphenopalatine artery met this problem. It has stood the test of time. Operating, in 1926, before a number of my peers, I was embarrassed by inadvertently cutting the sphenopalatine artery. A dry field was immediately flooded. A previously quiet patient began to become unruly. Fortunately the vessel was ligated. The work then proceeded smoothly and assurance was given that the artery could be successfully handled.

When, after due study, one has decided to obliterate the sphenoid sinus the procedure is as follows: The thin anterior wall of the sphenoid has been removed. One can look directly into the sphenoid at the bottom of the operative field. With a long sharp periosteal elevator the mucosa is raised from the nasopharyngeal wall under the floor of the sphenoid. This periosteum containing the sphenopalatine artery is separated from the bone for about 1 cm. The bone which has been denuded represents the anterior part of the sphenoid floor. This is taken away with forceps of strong design. A special needle, threaded at the point, is passed through the mucosa into the sphenoid cavity. The vessel is included in the knot that is tied by special ring "tyer" already used in tying the ethmoid vessels.

Instead of proceeding at once to the removal of the floor of the sphenoid an involved procedure has been found by experience to be required. Many sphenoids are 3 to 4, even 5 cm. deep, while the distance between the rostrum of the vomer and the lateral wall—the width of approach—is not, in some cases, more than one centimeter. It is physically impossible to cut out bone, often heavy, for that great distance through such a narrow approach. Also, if technically possible to cut such a narrow ditch, granulation tissue would soon grow across and obliterate it. This disappointing experience led me to develop the following technic.

If the floor is to be taken away, it is obvious that greater width must be found for approach. This necessary space may be gained by utilizing the other sphenoid. A submucous resection of the nasal septum is performed. If there is any septal deformity, the incision is made anteriorly far enough to include it. If there is no septal irregularity of importance, what I call "a posterior submucous resection" is done. The incision through the septal mucosa in that case is made over the posterior part of the vertical plate of the ethmoid



and vomer. The thin vertical ethmoid plate is easily penetrated by even a blunt elevator. The septal mucosa is dissected free from both sides of the septum. It is spread apart and removed from the front of both sphenoids. One sphenoid has already been entered, the other is now opened from between the layers of the septal mucosa. The back part of the bony septum is removed with strong forceps. Next the rostrum of the vomer and then the sphenoid septum are removed. Both sphenoids have now been converted into one cavity. The approach is limited on either side only by the bases of the pterygoid processes. Working through the facial incision, the flaccid septum is pushed aside and the mucosa of the fornix of the nasopharynx is elevated from the sphenoid bone as far as this makes the floor of the sphenoid sinus. There is now ample room to remove the entire bony floor of both sphenoids. No floor whatever should be left to interfere with perfect downward drainage if obstinate cases are to be cured. After the bony floor has been removed, or while doing so, the mucosa is cut away underneath on the side on which the sphenopalatine has been tied. Experience has proven that it is not necessary to tie the opposite sphenopalatine artery nor resect the nasopharyngeal mucosa on that side. The drainage from that sphenoid is down over the intact mucosa.

My own experience in this connection has been illuminating. Impelled by the same humanitarian motive that led the kind gentleman to cut off his dog's tail an inch at a time, in a certain case, it took me four attempts to remove a very thick sphenoid floor. Until all the floor was removed there was recurrence of a purulent granulating mass. The floor once completely removed, the mucosa healed smooth and clean without crusts.

The fronto-ethmo-sphenoid operation as here briefly outlined is admittedly subject to fair adverse criticism. We can state in its favor that it represents a more thorough practical technic than has heretofore been presented. We can also claim a much greater percentage of so-called cures than by any other method. It has taken advantage of technic advanced gradually through the years. To its discredit we must admit that it does not always cure. Most of the trouble comes from the frontal sinus. The ethmoids and the sphenoids are obliterated by this technic. The frontal sinus is not obliterated. Seldom is the general surgeon asked to cure an abscess without obliterating the abscess cavity. The tuberculous abscess in the lung and the pleural empyema have both at last been met by the surgical principle recognizing the necessity for the obliteration of the abscess cavity. The dentist at last has abandoned the abscessed tooth. The rhino-

logical surgeon, however, is asked to handle the abscessed frontal and maxillary sinuses without obliteration of their cavities. Our only chance for cure, therefore, depends upon adequate and permanent dependent drainage, not simply "aeration." Satisfactory drainage downward through the exenterated ethmoid mass may be expected immediately following the technic outlined. Ordinarily this opening can be counted upon to satisfactorily combat the inflammatory conditions accompanying an acute sinusitis. A weakness, however, inherent in the principle employed, allows the wide frontonasal opening, left after operation, gradually to narrow and even close completely in a small percentage of cases. The removal of the floor of the frontal sinus, the frontal process of the maxillary bone and the lamina papyracea take the support from the soft parts overlying. They are gradually drawn inward by granulation and scar tissue until the immediately satisfactory postoperative frontonasal opening may be narrowed or completely closed.

Much thought and effort have been spent upon the problem of keeping this nasofrontal space open. The removal of the bony window is essential to entering the operative field. This includes the anterior part of the lamina papyracea. As this part of the lamina cannot be preserved, the rest of the lamina is perhaps best sacrificed. It makes the technic easier, allowing one to follow the ethmoid cells more easily toward the orbital apex. Also it eventually allows the orbital soft tissues to crowd over and encroach on the exenterated

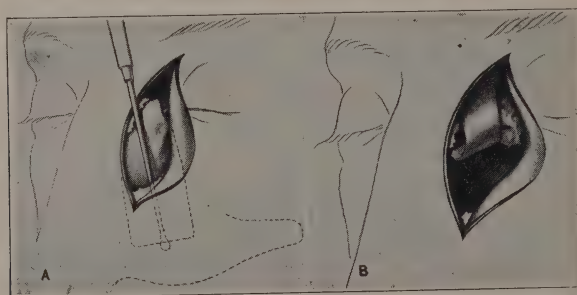


FIG. 1. *A*, the nasal mucosa has been bared. The separation of the flap and the approximate extent of flap are shown. *B*, the flap of nasal mucosa attached above is turned upward to line the frontonasal opening and the sinus.

ethmoid region. It thus reduces the nasal space advantageously. Many methods have been employed for keeping the frontonasal opening patulous. The long wearing of a rubber tube, dilatation by sounds, skin and mucous membrane grafts, have all been used with varying success and failure. When the frontonasal opening closes,

drainage from the frontal sinus stops. Unless the diseased cavity has become obliterated by new formed tissue trouble is to follow.

To prevent the disastrous closure of the frontonasal opening I have developed a flap. This is made of the nasal mucosa, principally from the agger region of the nose.

*The Frontonasal Flap.*—The frontonasal flap is formed and disposed as follows: Working through the facial incision and bony window, the nasal mucosa, which has been carefully preserved, is separated from the nasal process of the maxillary bone. This is accomplished by passing variously curved periosteal elevators downward. The mucosa can be elevated well down, even off the medial surface of the inferior turbinate. Working from the anterior naris, a horizontal incision through the nasal mucosa down upon the underlying elevator outlines the lower free border of the flap. Vertical incisions accomplished through the naris and through the external incision complete the formation of a quadrilateral flap, free below and attached at the roof of the nasal fissure. This sturdy piece of mucosa from 1 to 3 cm. in width and from 3 to 6 cm. in length is turned upward to line the medial wall of the frontonasal opening and more or less of the frontal sinus. After months of experimentation with this flap and the ethmoid flap to be described I was hesitant, working on the cadaver, because of the friability of the embalmed mucosa. Fortunately the interest of the Los Angeles study group, before whom

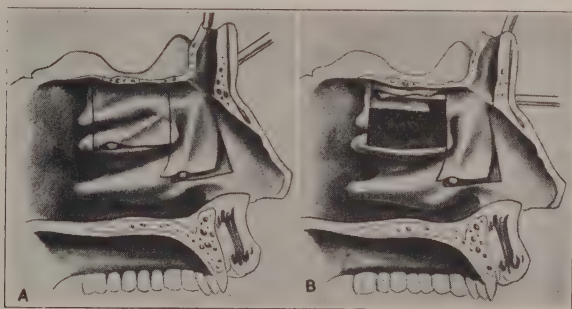


FIG. 2. A, the preparation of the two flaps is shown from the nasal aspect. B, the ethmoid flap is being smoothed over the denuded surface of the ethmoid roof. The frontal flap is still represented in the nose.

I operated in January, 1934, induced me to try these modifications on the living. I found the mucosa tough enough for the necessary manipulations and the technic practical. I now have been using the flap for a year and am much encouraged by the fact that none of the openings into the frontal sinus have closed. It puts the operation for me on an entirely new footing.

*The Ethmoid Flap.*—The ethmoid mucosa covering the medial aspect of the ethmoid mass is now disposed of as follows: All the thin bone, including the medial plate of the superior and middle turbinates, is carefully removed from this mucosa. This sheet of mucosa is plastered over the ceiling of the ethmoid mass, *i.e.*, the inferior surface of the dural plate covering the lateral mass of the ethmoids. The median bony wall of the ethmoid mass has been trimmed down almost level with the cribriform plate. This can be done with perfect safety with sharp-cutting bone forceps, working with perfect vision in a bloodless field. The mucosal flap covers the denuded bone from the cribriform plate laterally.

A flap similar to this was described by Monson in the *Archives of Otolaryngology* for January, 1934. This is ingeniously conceived by the author for use with the intranasal technic. My ethmoid flap for use with the external technic was developed after a long period of experimentation and applied before this article reached me. I believe the principle will find its greater field of usefulness in association with the external operative approach. The necessarily incomplete nature of the work done by intranasal ethmoid technic has been too well recognized to need emphasis. In view of that fact one might fear the sealing of partly exenterated ethmoid spaces.



MEETING OF THE MID-WESTERN SECTION HELD IN  
DALLAS, TEXAS, ON JANUARY 28 AND 29, 1935,  
UNDER THE CHAIRMANSHIP OF DR. EDWARD H.  
CARY OF DALLAS, TEXAS.

AUTOPLASTIC NERVE GRAFTS IN FACIAL PALSY

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The objects of this paper are two: first, to pay a humble tribute to the work of Sir Charles Ballance and Dr. Arthur B. Duel in establishing experimentally and practically that the restoration of continuity of the facial nerve is the rational and only thoroughly satisfactory means of dealing with facial palsy in all but exceptional cases; second, to call attention to the fact that this work is possible to any otologic surgeon of fair skill who will master the details of the technic of the operation and the after-care of the wound.

The report of the work of Ballance and Duel<sup>1</sup> represents to me one of the greatest contributions to clinical research in modern times. I have read this publication many times, and each time I get something more from it and am impressed anew by the thoroughness of the experimental work, the true scientific spirit displayed and the soundness of the conclusions reached. In the later addresses of Duel before the American Laryngological, Rhinological and Otological Society<sup>2</sup> and the Royal College of Surgeons of England<sup>3</sup> the progress of the practical application of the results of the research is outlined in a series of cases. In a recent personal letter he informs me that he has now operated on eighty-five cases.

Not less noteworthy and praiseworthy in these reports is the thorough and painstaking detail with which is described every step in the operative technic and the after-care of these cases. This is in marked contrast with the practice of many surgeons who describe the beautiful results obtained by their methods but are so vague in the description of operative technic as to encourage the suspicion that they are deliberately concealing something.

I shall not burden you with a review of the work of Ballance and Duel. Owing to the fact, however, that for years so many of us had the fixed idea that anastomosis operations afforded a satisfactory solution of the problem, I am taking the liberty of quoting the following conclusions from them: "There can be at the present time no question as to the disability attending an anastomosis operation, and as to the imperfections present in many cases that were hailed as

good recoveries in the past. In further considering the future of the operation for the relief and cure of facial palsy, we are driven to the conclusion that the anastomosis operations of the past may be looked on as a stage in the evolution of this branch of surgery." Again, "By exposing and dealing directly with the damaged portion of the nerve, the surgeon's efforts will result in a better recovery; no associated movements or paralysis of muscles subserving other regions will mar recovery. We hold strongly that anastomosis operations for the cure of facial palsy should be banished from future surgical practice. Nevertheless, to every law there are exceptions. When the facial nerve in all its long course within the fallopian canal has been destroyed, for example, by necrosis of the petrous or by gunshot wound, so that an intratemporal nerve grafting operation is not possible, there is nothing left to do but to perform the old operation of anastomosis."

I may say, in passing, that here Ballance ignores completely the efforts of plastic surgeons, notably Sheehan of New York and Gillies of London, to use muscle and facial transplants to correct the deformity and restore a modicum of movement to the face.

Another fact was brought out by the work of these investigators which is of no less importance from a clinical standpoint than the autoplasmic nerve graft operation. I refer to the conclusion that when facial palsy develops, either spontaneously or due to operative trauma, if the nerve is exposed and the sheath incised, the necessity of further operative procedures will be obviated and partial recoveries prevented.

#### REPORT OF CASES

*Case 1.*—V. W., age twenty-two, consulted me June 8, 1933, with severe pain in his right ear. He had been suffering from an infection of his nose and throat for two weeks and the ear had been affected for two days. Upon examination the tympanic membrane was red and bulging. On incision a copious serous discharge escaped. He was seen the following day, when all subjective symptoms were relieved and the ear discharging freely. He did not return until June 29, three weeks later. At this time he had a very frank case of mastoiditis, as evidenced by a very copious, thick, purulent discharge from the ear, sagging of the posterosuperior canal wall, tenderness over the whole mastoid process, edema and special tenderness over the mastoid tip, a dull headache and a temperature of 100° F. X-ray examination showed breaking down of the trabeculae. Operation was advised at once but delayed by the patient for four days.

On July 3, 1933, the mastoid was opened under ether anesthesia. The cortex was thin and a large pneumatic process was found, full of pus and large granulations. The whole tip was broken down and all of the cell walls soft and easily removed with a curette. The process was thoroughly cleaned out to the inner table and the wound closed with a rubber tissue drain.

The patient awoke from the anesthetic promptly and showed no untoward symptoms during the day, but during that night developed a paralysis of the right side of the face. The nurse thought that when she first noticed it the loss of function was only partial, but the next morning it was complete.

Since the palsy was not present until several hours after operation I thought it was due to postoperative edema and would clear up in a short time, as I had seen in cases of late palsy coming on after radical operations.

The healing of the mastoid wound was uneventful. When the facial palsy showed no sign of improvement I suggested reopening the wound, but the patient did not want it done until we were sure it would not clear up spontaneously. He reported back occasionally for observation and when no improvement was noted after three months, operation on the nerve was urged. He consented, but for various reasons delayed it until March 12, 1934, eight months after the onset of the palsy.

**Operation:** Under ether anesthesia the mastoid was reopened and a radical operation performed. Flaps were cut from the canal and sutured back. The posterior canal wall was gradually lowered until the facial nerve was exposed. About half way between the knee and the stylomastoid foramen a hiatus in the nerve, almost a centimeter in length, was found. The nerve at either end of the gap was enlarged and bulbous. The nerve was well exposed from the knee to just above the stylomastoid foramen, the bulbous ends were trimmed off somewhat and two strands of the internal cutaneous nerve, removed from the forearm, placed in the bony groove so as to fill snugly the hiatus in the facial. The whole exposed nerve was covered with a strip of gold foil and the wound loosely packed with short strips of gauze wet with normal salt solution. The packing was not disturbed for three days, but was kept moist with saline solution. After three days the dressing was changed daily, great care being exercised not to disturb the gold foil covering the nerve. At the end of two weeks the foil was carefully removed and the nerve found entirely covered with healthy, pink granulations. The patient was dismissed from the hospital and returned to the office for dressings. Healing was uneventful. Epidermitization was complete in eight weeks. The posterior wound was not closed by a secondary operation for the reason that it looked as if it would close spontaneously. It did not, however, and he has a postauricular opening which he has thus far refused to have closed.

**Subsequent history:** The patient reported back at fairly regular intervals for observation. For nearly five months there was no change, subjectively or objectively. In August he reported that he could distinguish a difference. The face felt as if it wanted to move and he had a tingling sensation in it at times. No change was apparent objectively. From that time, however, each month there was a visible improvement in the tone of his facial muscles. On November 9 there was some voluntary movement of his lower lid and his face was practically straight in repose. He said that people no longer noticed that there was anything abnormal in his appearance and asked him about it as they did almost daily before. He stated that his face felt as if it were moving and that he often went to the mirror to see whether or not it did, but he had been unable to detect any motion. On November 21 he called up in great excitement to say that he could move his face. He came to the office the next day and he could raise the angle of his mouth and close his eye fairly well. This power of movement was not constant. At times he could exercise it fairly well and

again scarcely at all. There was no motion in the occipitofrontalis and his face was markedly asymmetrical on smiling.

During the next two months improvement was gradual, but slower than I hoped for. He still cannot elevate his forehead. He can smile symmetrically, but on laughing the face is drawn to the left to some extent.

Whether or not this case will proceed to complete restoration time alone will tell. I shall watch it with interest and be glad to report further at a later date. It is evident that the nerve has grown through the graft to the face. The explanation of failure of the frontalis to move may be the longer distance for the nerve to go, the failure of certain axons of the nerve to grow out, or to fibrosis of the muscle. If no further improvement occurs the patient will be satisfied.

*Case 2.*—Theresa M., aged five years, had an operation on July 31, 1934, for acute mastoiditis with a subperiosteal abscess on the left side. The wound continued to discharge for three months, and she was admitted to the Houston Eye, Ear, Nose and Throat Hospital on November 11, 1934, under the care of one of my associates, Dr. J. M. Robison, for a secondary operation.

The mastoid wound was reopened and extensive involvement of the bone found extending into the zygoma and into the bone surrounding the semicircular canals. The softened bone extended very deep, and there was found an exposure of the facial nerve in its descending portion. I was not present at the operation, but a few hours afterwards Doctor Robison informed me of the findings and the fact that a complete paralysis of the left side of the face was found as soon as the child awoke from the anesthetic. At my suggestion the child was taken back to the operating room and the wound reopened. The facial nerve was found exposed for a distance of almost a centimeter midway of the descending portion. It was dark blue in color, looking more like a vein than a nerve, and had a boggy appearance. The bone above and below the exposure was removed for some distance and the sheath of the nerve incised with a fine knife. The nerve was carefully covered and the wound loosely packed with gauze saturated with normal sodium chloride solution.

Subsequent history: The nerve quickly became covered with granulations. In five weeks some motion of the facial muscles was evident and from that time the progress of recovery was rapid. Nine weeks after operation no evidence of palsy could be noted.

#### COMMENTS AND CONCLUSIONS

1. Cause of the palsy. A careful study of Case 1 convinces me that the only rational explanation of the sequence of events is that at the time of the mastoid operation a large pneumatic mastoid was found; the cellular structure was badly broken down; a cell extended to the fallopian canal, and in scraping it out the sheath of the nerve was injured; following this trauma the nerve swelled and paralysis developed. Due to this swelling, and possibly infection, the portion of the nerve exposed sloughed out. The nerve could not have been severed or paralysis would have been immediate.



In Case 2 the nerve was probably traumatized a little more severely, as paralysis came on at once. The appearance of the nerve gave evidence that it was already affected, as it was blue and unhealthy in appearance.

I am convinced that the prompt interference in this case prevented the development of the same condition that was seen in Case 1. I feel equally sure that the loss of nerve tissue and the necessity for the autoplasmic graft operation in Case 1 could have been avoided if we had handled this case as we did Case 2.

It may be argued that the second case might have cleared up promptly if let alone. Of course it is impossible to prove the point, but the nerve was tightly compressed by the bony canal at either end of the exposure, was swollen, blue and unhealthy looking. It would have been but a step to gangrene and a slough. Be that as it may, the procedure adopted was easy of execution and the result most satisfactory. If I am so unfortunate as to have another case of facial palsy following operation I shall certainly advise immediate exploration. The only exception I should be inclined to make is in a case coming on several days after a radical mastoid operation. I have never had an immediate facial palsy in connection with the radical operation, but have had several, partial or complete, coming on from two to seven days after operation. These have all cleared up completely in a short time and this experience naturally makes me optimistic in this particular type of case.

2. Causes of delay in return of function. (a) Delay in operation; eight months elapsed after the onset of the palsy. It is probable that by that time changes had occurred in the muscles. (b) The use of a fresh graft instead of one in which Wallerian degeneration had taken place. It was not until two weeks after I did this operation that I learned of the new technic advocated by Duel, by means of which he finds the time for recovery is cut to from a half to a third of the time required where a fresh graft is used. (c) I believe that if I had removed all of the enlarged ends of the nerves and had slit the sheath beyond it, it is possible that the outgrowth of axons from the central portion and ultimate restoration of function might have been hastened.

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# THE CLINICAL RELATIONSHIP OF INFECTIONS IN THE UPPER RESPIRATORY TRACT TO CERTAIN TYPES OF CHRONIC UVEITIS

(A PRELIMINARY REPORT)

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Since Rosenow and Billings first pointed the way to a clearer understanding of the relationship between chronic focal infection and the eye, it has become generally accepted that such foci are pre-eminently the cause of certain inflammatory diseases in that organ. The location and duration of the responsible foci are variable, but they have been proven to have a direct causative bearing on such chronic inflammatory reactions by numerous clinical investigators. So far as the eye is concerned the vascular tunic, or the uveal tract, is predominantly the area involved in these so-called elective localizations. Foci in the upper portion of the respiratory tract are of paramount importance in the causation of chronic uveitis, as they are the greatest in frequency of all foci encountered in a search for the underlying cause.

Uveitis is a term used to designate inflammation of the vascular tunic of the eye and is divided clinically into an anterior type in which the iris shows the principal involvement, and a posterior type in which the ciliary body and choroid show the principal changes. Ofttimes there is an extension of the pathologic change from one part of the uveal tract to another, because of the close anatomic relationship which exists between them.

Frequently uveitis is considered as a disease which is manifested only by certain classical changes in the anterior segment of the globe, such as deposits on the corneal endothelium, the so-called keratitis punctata, injection of the ciliary blood vessels, inflammatory changes in the iris, and oftentimes coincident alteration in the clarity of the fluid media.

The type of uveitis with which we are concerned is the low grade insidious type involving the vascular structures back in the iris, the so-called posterior type of uveitis, in which the onset is of a relatively painless nature, accompanied by changes in the consistency of the vitreous which becomes abnormally fluid. Floating opacities in the vitreous, which vary in degree from spider-web strands to veritable sheets of exudate, asthenopic symptoms, photopsiæ and re-

duction in visual acuity are also observed. Quite often there is associated tenderness of the eyeball when it is palpated, but marked pain is usually not encountered. Demonstrable changes in the choroid can oftentimes be made out if the opacity of the media is not too great. Extension to the more anterior or more superficial portions of the globe will often occur coincidentally or consecutively in chronic uveitis.

One hundred and twenty patients with chronic posterior uveitis due to focal infection which form the basis for this paper were subjected to a critical survey with a view to determining the relative frequency of various foci of infection which might have some etiologic bearing on the eye condition. Paranasal sinus infections were surprisingly frequent, occurring in fifty-eight patients, or 48.33 percent. Of the total number (120) forty-seven patients, or 39.16 percent had chronic infection in the ethmoid sinuses. In eleven patients, or 9.16 percent, the infection was localized in either one or both maxillary antra. There were no patients in which the frontal or sphenoid sinuses alone showed demonstrable infection. The usual type of infection encountered was the low grade inflammatory reaction which is usually designated as latent or concealed infection. In this series of patients there was only one instance of acute sinus inflammation occurring in conjunction with uveal disease, and that was an acute exacerbation in a chronically diseased frontal sinus. Reference to the accompanying table will show the incidence of foci of infection in other parts of the body.

The methods used in diagnosis of paranasal sinus infections were the same in all cases and were based on the usual objective findings, such as the location and character of the nasal discharge, gross appearance of the nasal tissues, cultural and cytologic examination of the discharge, the x-ray findings and transillumination where applicable. In quite a few instances operative verification and histologic examination of the excised soft and bony tissue was made. In the tissues examined the histologic changes were chiefly those of hyperplasia, with round cell infiltration, principally lymphocytic, and with an especially striking perivascular cuff of leucocytic infiltration which oftentimes extended into the bony tissue.

The average age of the patients was forty-six years, the eldest being eighty-six years and the youngest thirteen years. The sex distribution was about equal, there being sixty-six females and fifty-four males represented. The greatest number of cases occurred in the fourth and seventh decades of life.

Evidence of chronic tonsillar infection was present in sixty-one patients or 58.33 percent, the only area showing a higher percentage of involvement than the paranasal sinuses.

Some type of dental sepsis was present in thirty-four, or 28.33 percent, of the patients, which is in conformity with the observations of other writers on this subject.

Constipation is almost universally present in chronic posterior uveitis and it is of interest to note that thirty-one patients, or 25.9 percent of the total were suffering from chronic colitis. The colitis was almost invariable of the spastic type and associated with a change in the normal bacterial flora of the intestine. The hemolytic streptococcus was most frequently considered the offender, and it was surprising to find that quite often the same type of pathogenic organisms was present in both the stool culture and the culture obtained from the nasal or sinus discharge. It would appear from this investigation that patients suffering from chronic paranasal sinus disease are especially prone to acquire infection in the lower bowel by swallowing infected secretions over a long period of time and that this infection is most likely to be of the same type encountered in the nasal discharge. There was only one patient in the series in which Barger's bacillus was found in the stool culture. The organism was not present in the nasal discharge. The infection in the colon may often be considered a focus of infection secondary to the primary nasal focus.

In addition to the observations noted above, twenty-four patients were tested intracutaneously with uveal pigment to determine if they exhibited hypersensitiveness to uveal pigment. In all patients tested it was found that some degree of cutaneous hypersensitiveness existed. The usual control injections of 1:20 bovine serum were used and were negative in all cases. The latter cases were not included in the series. The degree of reaction induced by the intracutaneous injection of uveal pigment varied from mild local erythema, with slight induration about the point of injection, to more severe reactions characterized by induration with vesiculation and ulceration. In two patients constitutional reactions, such as nausea and vomiting, with rise of temperature, followed the skin test. In these tests we have used the so-called normal pigment suspension made from bovine eyes as well as autolyzed pigment obtained from the same source, but we have concluded that the normal uveal pigment suspension is most active as a test antigen.

Infection in one or more of the paranasal sinuses which continues for a variable period of time may reach a stage which we



designate as chronic, and in which the symptoms referable to the nose may be so mild that they escape the patient's attention entirely or make little impression. In our series of patients the duration of the antecedent sinus infection estimated from the case histories was slightly in excess of two years. It is in such mild sinus infections that elective localization is prone to occur in the vascular tunic of the eye. At the point where this blow is delivered a low grade inflammatory reaction is set up which calls forth a cellular response with infiltration in the area about the choroid vessels with the usual cells characteristic of such reactions. Liberation of various enzymes, proteolytic, saccharolytic and lipolytic, occurs when these cells disintegrate. It is most probable that through their action the protein-like uveal pigment, which is ordinarily nonabsorbable, is acted upon and converted into a soluble substance which is slowly absorbed and acts as an allergin which sensitizes the patient to uveal pigment. Once the patient is sensitized then secondary allergic reactions will be produced wherever a similar type of pigment occurs in the body. Naturally this means that the fellow eye is prone to become affected, and from a practical standpoint this is exactly what occurs, as a very high percentage of patients with chronic posterior uveitis have the disease in both eyes. If uveal pigment is introduced into the skin as a test antigen a reaction characteristic of hypersensitiveness will occur at the point of injection. This would indicate that the remote effect of local infection which electively localizes in the eye is the production of a special type of allergic reaction, namely, hypersensitiveness to uveal pigment.

Another type of reaction can, according to some observers, be produced through the action of toxins from the focus of infection on tissue of the uveal tract which has become previously sensitized to the organism elaborating it, and would explain the absence of bacteria in such eyes when they are subjected to histologic examination. This theory seems to be borne out in our cases which have been tested intracutaneously with vaccines made from organisms obtained from sinus discharges, as in the patients investigated in this manner hypersensitiveness to autogenous vaccine was almost universally present. Here it may be relevant to state that heat-killed bacterial suspensions are preferable for skin testing to avoid the confusing reactions which come from tricresol, as approximately one-fourth of all persons are hypersensitive to tricresol when introduced intracutaneously, even in weak dilutions.

It is interesting to note that pigment of type similar to uveal pigment occurs in the labyrinth of the internal ear, which may

partially explain the symptoms of vertigo which is so often complained of in ethmoid disease. Extension of the reactions from the labyrinth to the cochlea may explain the rather frequent association of deafness and uveal disease or offer an examination for such a condition as Ménière's disease on an allergic basis. Unfortunately our observations concerning the frequency of this condition did not include all the patients in the series, so the exact frequency of its occurrence cannot be stated, as the relationship was not clearly evident early in the series, but from the number of patients in the series that have been investigated from this standpoint we are inclined to believe that demonstrable impairment in hearing is present in well over one-fourth of the patients and that vertigo due to labyrinth irritation is equally frequent. Where labyrinth tests have been employed we have not found any definite diagnostic evidence of disease in this organ other than an occasional instance of hyperirritability, but a more extensive survey on this special point is contemplated in future cases.

The anterior portion of the uveal tract, the iris, apparently does not possess this capacity for producing sensitization to uveal pigment, which is probably accounted for in the fact that there is a structural or chemical difference in the type of pigment present in this structure and the ciliary body and choroid.

It is also of interest to note that fifteen patients, or 12.5 percent, of the total number had demonstrable cataractous changes in the crystalline lens at the time they came under observation because of their uveitis. It has been noted by practically all ophthalmologists that cataractous changes in the lens supervene with great frequency in patients with chronic posterior uveitis, and indicates most forcibly that cataractous lenticular changes are the terminal event in a chronic low grade inflammation of the vascular tunic of the eyeball which had its inception in a focal infection.

By far the most dangerous feature of uveitis is its relatively painless course, which may and often does influence the patient to postpone treatment until the condition has reached a stage where irreparable damage has been done. How fortunate then is a patient who has headaches or other annoying symptoms referable to his sinuses which force him to seek relief at an early stage of his illness. It is at such times that a thorough and painstaking examination would reveal ocular pathology when the greatest good could be accomplished by treatment.

So far as treatment of the underlying nasal infection is concerned, it is not considered within the province of this paper to

discuss this phase of the subject, as it would be foreign to the title under consideration, but it may be stated that all too frequently the various paranasal foci of infection cannot be cleared up entirely, and in such instances we must be content in minimizing the degree of infection present. The difficulty is greatest where extension into the surrounding bony tissue has given rise to a low grade osteitis of a type which cannot be greatly influenced by operation.

Oftentimes residual infection remains in the paranasal sinuses after they have been operated upon and necessitates careful post-operative treatment to eradicate it or minimize its effect.

This article is intended to correlate certain facts observed in a group of patients suffering from infections in the upper respiratory tract, particularly the paranasal sinuses, with coincident ocular dis-

TABLE 1

Total number of patients with chronic uveitis, 120.

Average age, 46; eldest patient, 86 years; youngest, 13 years.

Patients tested and treated with uveal pigment.....	24	20 percent
Patients showing infection in sinuses.....	58	48.33 percent
Patients having ethmoid sinusitis.....	47	39.16 percent
Other sinuses involved.....	11	9.16 percent
Patients showing positive tuberculin reaction.....	2	1.66 percent
Patients showing positive Wassermann.....	7	5.83 percent
Patients showing tonsillar foci.....	61	58.33 percent
Patients showing dental foci.....	34	28.33 percent

Occurrence of chronic posterior uveitis by decades:

<i>First</i>	<i>Second</i>	<i>Third</i>	<i>Fourth</i>	<i>Fifth</i>	<i>Sixth</i>	<i>Seventh</i>	<i>Eighth</i>	<i>Ninth</i>
0	7	13	26	20	19	26	7	2
1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90

TABLE 2

## COMPLICATIONS

	<i>Percent</i>		<i>Percent</i>
Kidney infection .....	6 or 5	Retinal detachment ....	3 or 2.50
Prostatic infection .....	3 or 2.50	Hay fever .....	2 or 1.66
Colon infection .....	31 or 25.9	Arterial hypertension...	5 or 4.16
Lenticular opacities .....	15 or 12.5	Episcleritis .....	1 or 0.833
Heterochromia iridis ....	3 or 2.50	Acute glaucoma .....	1 or 0.833
Ophthalmic migraine ....	3 or 2.50	Retinal hemorrhage ...	2 or 1.66
Pulmonary tuberculosis ...	2 or 1.66	Thrombosis, central	
Diabetes .....	2 or 1.66	retinal vein .....	1 or 0.833
Hypothyroidism .....	2 or 1.66		

(Multiple complications were not infrequently observed in the same patient.)

case, in the hope that such a critical survey would reveal facts which could be put to service in the relief of an unfortunate group of patients which constitutes one of the most tragic chapters within the limits of our specialty.

All patients included in this survey were subjected to the usual serologic tests to exclude syphilis, which was found to be present in only seven, or 5.83 percent, of the total number. In the 120 patients constituting our series there were two patients exhibiting hypersensitiveness to tuberculin, and in those patients there was definite pulmonary tuberculosis, which was not, however, considered positive evidence that their ocular pathology was of a tuberculous nature. This is striking, in view of the fact that the older observers were prone to ascribe such conditions as uveitis to either syphilis or tuberculosis.

There were forty cases in which the uveitis was unilateral and eighty cases in which it was bilateral. The right eye was affected in seventeen instances and the left in twenty-three, in the unilateral cases.

There have been no animal experiments in our work, but there is abundant evidence in the literature of the experimental production of uveitis from toxins, as well as bacteria and foreign protein to substantiate the hypothesis upon which this investigation is based.



## LYMPHATIC DRAINAGE OF HEAD AND NECK— EMPHASIZING SPECIAL STRUCTURES\*

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All the structures of the head and neck have their lymphatic drainage either directly or indirectly into the superficial and deep cervical lymph glands. The superficial cervical glands are arranged along the course of the external jugular vein and its tributaries and around the superficial structures of the anterior triangle of the neck. The efferent vessels of these glands drain the lymph into the deep cervical glands. Since we are concerned chiefly in this discussion with the lymphatic drainage of the deeper structures of the head and neck, a detailed description of the superficial glands will not be necessary. However, special groups of superficial glands will be described where they are concerned with the drainage of certain structures. The deep cervical glands are arranged along the carotid vessels and are divided into superior and inferior groups.

The superior group includes all those glands related to the carotid arteries which lie above the omohyoid muscle. We may recognize a medial group, situated in front of the carotid vessels, and a lateral group, situated behind those vessels. The upper medial group receives afferent vessels from the nose, paranasal sinuses, face, mouth, tongue, ear, tonsil, upper parts of the pharynx and larynx, upper part of the thyroid gland, submaxillary, sublingual and parotid salivary glands, and from the interior of the cranium. The efferents from these glands pass both to the inferior deep cervical glands and to the jugular trunk. The upper lateral group receives lymph from the ear, the posterior part of the scalp, the superficial cervical glands, the brain, and the suboccipital region. These glands are intimately connected with the medial group, and their efferents pass to the inferior deep cervical glands and to the jugular trunk (Fig. 1).

The inferior deep cervical lymph glands (supraclavicular) lie below the omohyoid muscle and are divided into a medial and a lateral group. The medial group is closely related to the terminal portion of the internal jugular vein. These glands receive afferents from the upper deep groups, from the pretracheal and paratracheal glands, and from the upper part of the thorax. The lateral inferior cervical glands

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lie in the subclavian triangle, over the third part of the subclavian artery. They receive afferents from the lower parts of the neck, larynx, pharynx, thyroid gland, upper part of the trachea, the upper part of the thorax, and from the deep portion of the mammary gland.

The lymph vessels from the external ear and meatus pass to the anterior and posterior auricular glands, which are situated immediately anterior and posterior to the auricle, respectively, and these

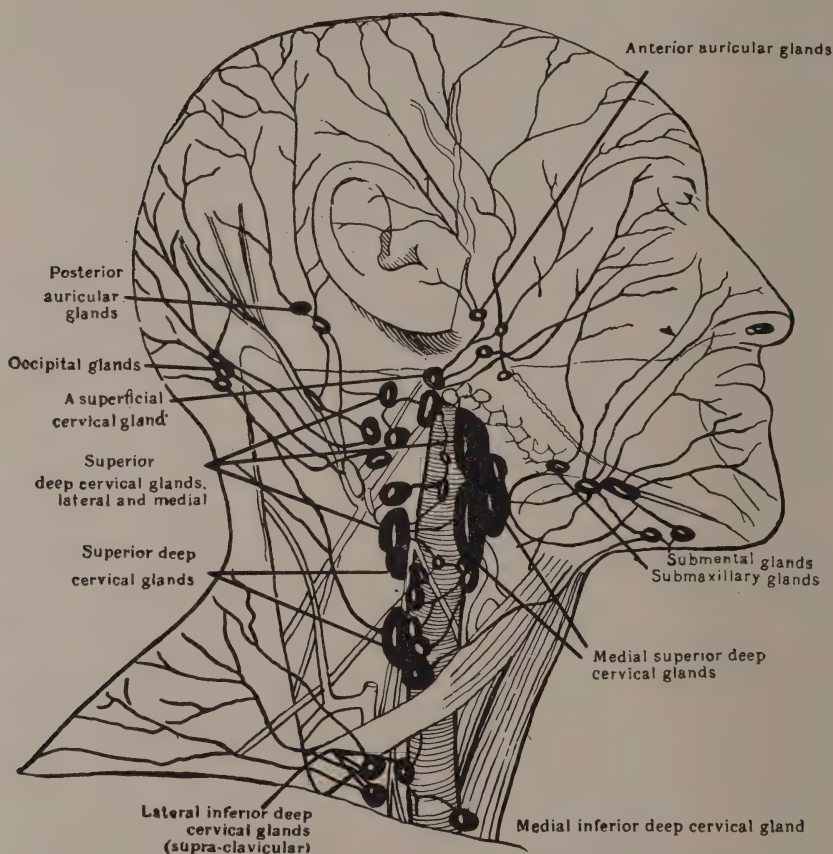


FIG. 1. Lateral view of the head and neck showing the general arrangement of the lymphatic glands and ducts.

glands send efferents to the superficial and superior deep cervical glands. The efferents from the lateral parts of the middle ear pass to the posterior auricular glands, and from the medial part of the middle ear and auditory tube they pass to the retropharyngeal glands. The retropharyngeal group consists of a number of glands situated in the loose connective tissue lying between the posterior wall of the

pharynx and the superior portion of the prevertebral fascia (Fig. 2). Both of these groups send efferents to the superior deep cervical glands.

The lymph vessels of the external nose form two groups, namely, superior and inferior. The upper group of ducts ends in the anterior auricular glands, and from here efferents pass to the parotid and upper deep cervical glands. The inferior group of vessels from the nose passes to the submaxillary lymph glands, and these glands in turn send efferents to the superior deep cervical glands. The lymph vessels of the upper and lower parts of the nose communicate with one another to a certain degree but not freely. The lymph vessels

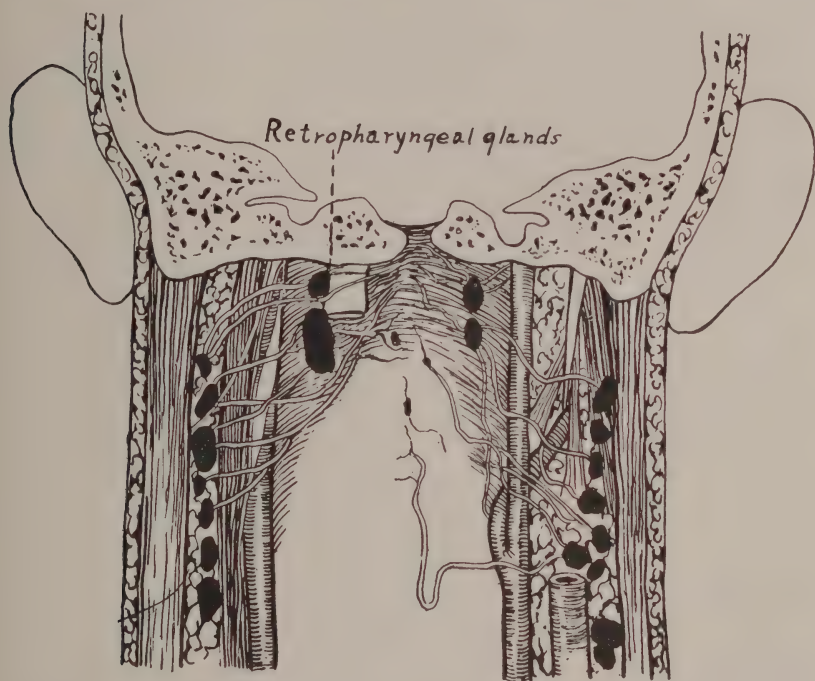


FIG. 2. Diagram of the retropharyngeal lymph glands.

from the anterior part of the interior of the nose pass to the submaxillary glands, and those from the posterior part go to the retropharyngeal and medial superior deep cervical glands. The submaxillary glands are found in the submaxillary triangle immediately below the body of the mandible and in close relation to the submaxillary salivary gland. The retropharyngeal and submaxillary glands drain into the superior deep cervical glands. The lymph drainage of the paranasal sinuses is not definitely known, but it has been suggested

that vessels from these structures pass, like those from the nose, into the superior deep cervical and retropharyngeal glands.

The lymph vessels from the medial part of the lower lip pass to the submental glands and occasionally directly to the superior deep cervical glands. The submental group consists of two or three small glands lying in the superficial structures directly below the symphysis of the mandible (Fig. 3). Efferents from the submental glands terminate in the upper deep cervical glands. Efferents from the remain-

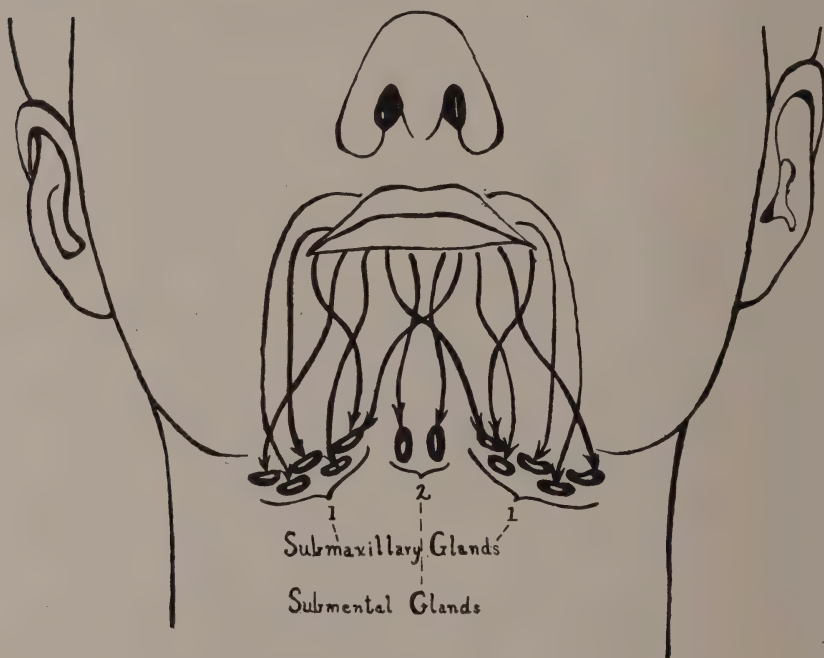


FIG. 3. Diagram of the lymphatic drainage of the lips.

ing parts of the lower lip unite with those from the upper lip and pass to the submaxillary lymph glands. From here lymph goes to the upper deep cervical glands. Some of the lip vessels may pass into the superficial cervical glands.

The lymph vessels from the cheek terminate in the submaxillary and superficial and superior deep cervical lymph glands.

Lymph vessels from the outer part of the anterior portion of the mandibular gum end in the submental glands. Those from the posterior part, together with those from the outer part of the maxillary gum, pass to the submaxillary glands. The vessels from the inner part of the gum of the mandible end in the submaxillary glands,



while those from the inner part of the gum of the maxilla and the hard and soft palates end in the medial superior deep cervical glands.

It is probable that the lymph vessels from the teeth of the mandible and mandible itself end in the submaxillary glands, and those from the teeth of the maxilla pass to the infraorbital glands, although this is not definitely known. Efferents from the latter group of glands pass to the anterior auricular and submaxillary glands.

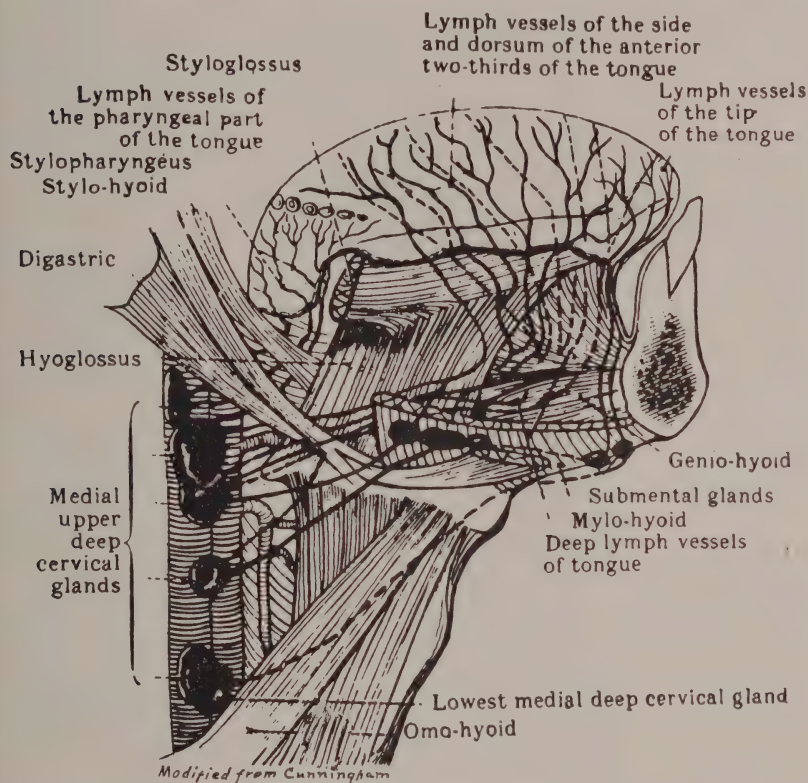


FIG. 4. Diagram of a lateral view of the tongue showing the arrangement of the lymphatic ducts.

The lymph vessels of the tongue are arranged in four groups— anterior, middle, posterior and deep. The anterior and middle groups communicate with each other and with those of the opposite side. The anterior group drains the tip of the tongue and the lower free portion and passes to the submental glands. The middle group drains the anterior two-thirds of the tongue, except the tip, and terminates in the submaxillary and superior deep cervical groups. The posterior vessels of the tongue drain that part of the tongue posterior to the

vallate papillæ and end in the superior deep cervical group. Vessels from the deep central part of the tongue go to the superior deep cervical glands (Figs. 4, 5).

The lymph vessels of the parotid gland end in the parotid and upper deep cervical glands. Vessels from the submaxillary salivary

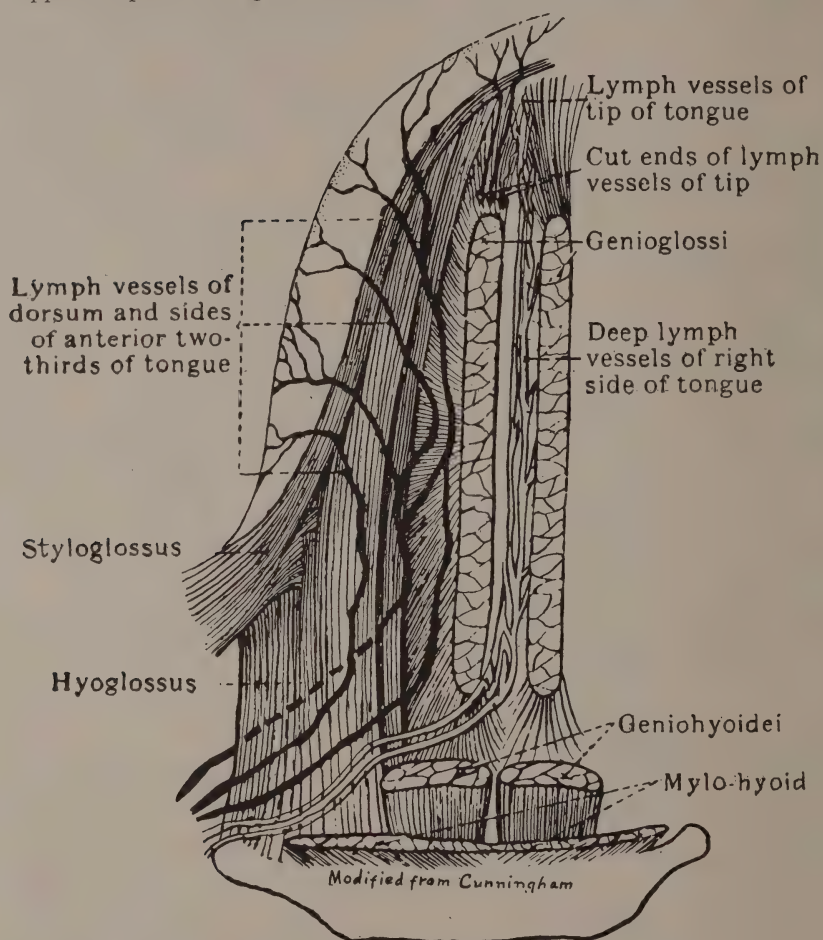


FIG. 5. Diagram showing the deep lymphatics of the tongue.

gland terminate in the superior deep cervical glands and not in the submaxillary glands as might be expected. It has not been definitely ascertained to what groups of glands the lymphatics of the sublingual glands pass; but it has been assumed that many of them join the vessels from the tip and free portion of the tongue, while others pass directly to the submaxillary glands.

The vessels from the upper and lateral parts of the pharynx drain into the retropharyngeal glands. Those from the anterior and lower parts of the pharynx pass to the upper medial group of cervical glands.

Lymph vessels from the palatine tonsil and the adjacent parts of the pharynx pass to the medial upper deep cervical glands. One gland, termed the tonsillar gland, which lies over the common facial vein in the digastric triangle, receives lymphatics directly from the palatine tonsil. Its efferents end in the medial group of superior deep cervical glands. This is the first gland that can be palpated in cases of acute tonsillitis.

The lymph vessels which drain those portions of the larynx above the vocal folds pass to the upper deep cervical glands. The vessels from the anterior part of the lower portion of the larynx pass through the cricothyroid ligament and end in the laryngeal, pretracheal and upper deep cervical glands, and these glands drain chiefly into the lateral group of inferior deep cervical glands. The efferents from the posterior part of the larynx terminate in the retropharyngeal glands. The lymph vessels of the upper and lower parts of the larynx communicate very sparingly over the vocal cords, but fairly freely in the posterior wall of the pharynx. This arrangement of the lymphatics of the larynx accounts for the edematous condition frequently encountered in the upper part of the larynx while the lower part of that structure remains uninvolved.

Although, strictly speaking, only the superior portion of the trachea is a neck structure, yet the lymphatics of the entire trachea and its subdivisions, the bronchi, are so intimately connected with one another that it does not seem out of order to give at this point a brief description of the lymphatics of all these structures. This discussion is based chiefly on the description given in Cunningham's Textbook of Anatomy, sixth edition.

The pulmonary lymphatic glands are found in the substance of the lung, between the bifurcations of the smaller divisions of the bronchi. These glands receive afferents from the lung substance and send efferents to the bronchopulmonary glands.

There are two groups of bronchopulmonary glands, situated one each in the hilus of the lung and around the bifurcations of the bronchi. They receive lymph from the lung substance, the pleura and from the bronchopulmonary glands. Their efferents pass to the tracheobronchial and intertracheobronchial glands (Fig. 6).

The intertracheobronchial glands lie below the trachea and in the angle formed by the trachea and the two bronchi. These glands

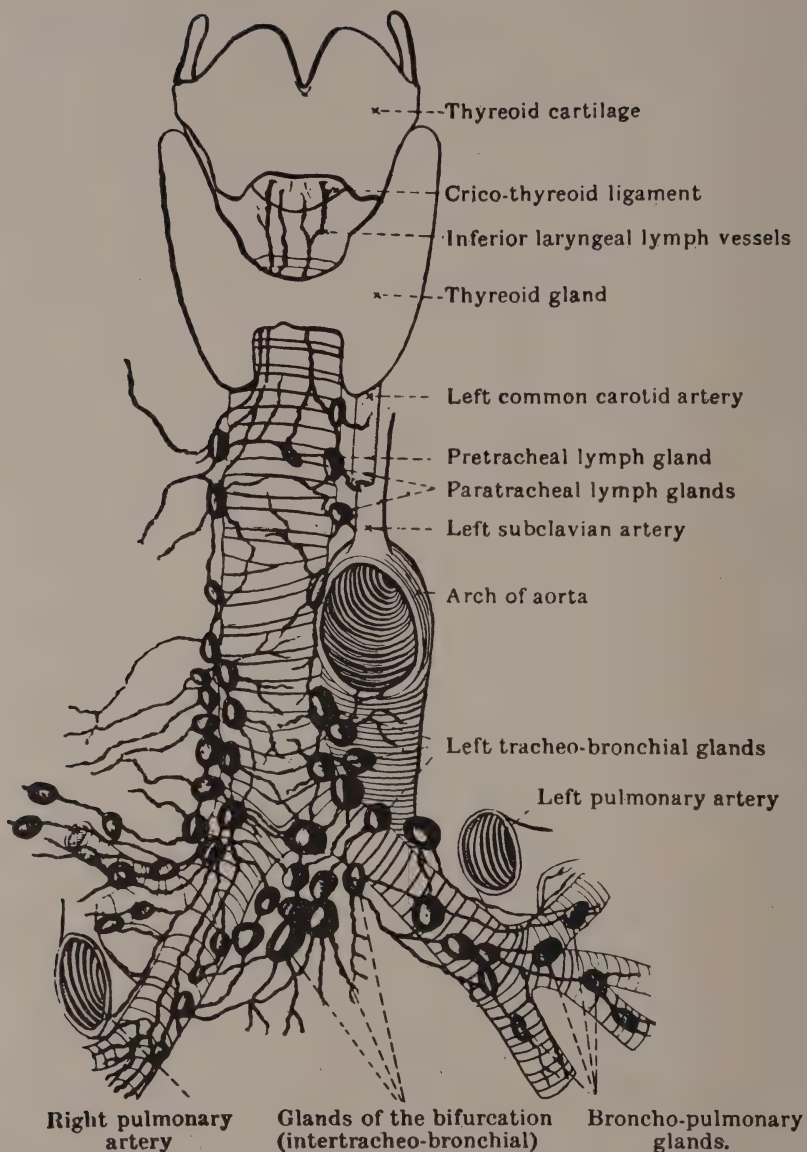


FIG. 6. Diagram of the lymph glands of the trachea and bronchi (modified from Cunningham).

receive vessels from the bronchopulmonary glands of both sides, from the heart and adjacent structures, and from the posterior mediastinal lymph glands. The efferent vessels of these glands terminate in the tracheobronchial glands of each side.



The tracheobronchial glands lie in the angle between the trachea and bronchus of each side. Lymph is received by these glands from the other bronchial glands, from the trachea and bronchi, and from the heart and mediastinum. The efferent vessels of the left group of glands enter the thoracic duct and communicate with the left medial inferior deep cervical glands. The right group of glands drains chiefly into the right bronchomediastinal trunk, and this duct joins either the right innominate vein or the right lymphatic duct. The bronchomediastinal glands of the two sides are intimately connected with one another and with the paratracheal glands.

The paratracheal glands lie on either side of the trachea and are connected over the surface of the trachea with the pretracheal glands. The paratracheal and pretracheal glands drain the upper part of the trachea, a portion of the thyroid gland and the lower compartment of the larynx. Their efferents pass to the tracheobronchial glands and to the inferior deep cervical glands.

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# THE CURABILITY OF MALIGNANT TUMORS OF THE UPPER JAW AND ANTRUM

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In the last two decades there has been marked improvement both in the treatment of malignant tumors of the upper jaw and antrum, and in the end-results. Twenty years ago, resection of the upper jaw was performed in such cases; some patients died following operation, and a small number of patients remained well five or more years after operation. In 1920, one of us (New) reported a method of treatment of malignant tumors of the upper jaw and antrum by means of cautery and irradiation, bringing out the fact that by this method of treatment operative mortality had been eliminated and the number of recurrences had been greatly reduced. Six years later, a second report was made of the end-results from treatment of ninety-seven patients with malignant tumors of the upper jaw and antrum by means of surgical diathermy, cautery, and radium, and the fact was again emphasized that the end-results revealed that more patients remained well by this method of treatment than by methods previously used.

## REVIEW OF LITERATURE

The literature on this subject recently has been very carefully reviewed in a monograph by Öhngren. He traced the development of electrosurgery and irradiation in the treatment of the condition on the Holmgren service of the Sabbatsberg Clinic at Stockholm. At first, the "soldering iron," or "ferrum candens," was used; large electric cauteries were then developed and, finally, electrosurgery. Öhngren reviewed both the foreign and American literature, and his monograph is an outstanding contribution to the subject. He has evolved a clinical classification of his own for determining malignancy of the tumor, in which he combines the position of the growth, with regard to whether indications for operation are favorable or unfavorable, its activity, determined microscopically, and the presence or absence of metastasis. He divides the skull, by means of an imaginary plane passing through the inner canthi and angles of the mandible, into an anterior-inferior or less dangerous portion and into a posterior-superior or more dangerous portion. In turn, each of these portions is further divided into four regions, two lateral and two medial.

Tumors in the medial posterior-superior regions are much more dangerous and treatment is more likely to result in a smaller percentage of cures than with those in the lateral anterior-inferior regions.

In England, the method of treating these tumors also has undergone evolution, as is indicated by two symposiums in the Section of Laryngology of the Royal Society of Medicine in 1922 and in 1931. In the first discussion the consensus was that these tumors should be removed by surgical cutting methods, the operative technic was stressed, and the method of using cautery and diathermy was thought little of by practically all the members. In November, 1931, however, the discussion revealed that the values of diathermy and of irradiation appreciated, and that resection by means of knife and scissors was little used.

A similar change in the treatment of these tumors has taken place in this country, as is indicated in articles by Sharp, by Quick, and by Houser. Most rhinologists, however, make an opening into the antrum for purposes of drainage only, and they do not attempt to destroy the tumor, when that is advisable, with surgical diathermy.

#### DIAGNOSIS

A diagnosis of epithelioma of the upper jaw, when the growth originates in the mucous membrane, is of course, not a difficult one. At times the condition is associated with papillary leukoplakia, a condition which potentially is malignant, and should be treated as such. Adenocarcinoma of mixed tumor type usually occurs posteriorly in the upper jaw and palate and, as a rule, presents a non-ulcerated, smooth hard surface. Fresh, frozen sections should be used in all cases to make a diagnosis at the time of operation, in order to determine the type of malignancy and its activity.

Patients with a constant complaint of some pain or uneasiness in the upper jaw or in the face, whose symptoms have been of short duration and who state that the pain or uneasiness has been made worse by lying down, should be carefully examined to exclude malignant tumor as the cause of the pain. Roentgenograms should be made and careful study carried out to exclude any involvement of bone. In the indeterminate cases, with symptoms indicating a possible tumor of the antrum, it is advisable to explore the antrum above the alveolar process in order to make sure that the patient's pain is not due to a malignant condition in the antrum. This exploration should be carried out by the surgeon who is equipped to take care of the condition if it is found to be malignant, thus avoiding the necessity for two separate operations, first, exploration and biopsy and then, later, a

second operation in which the malignant condition is cared for. Trauma associated with such a preliminary exploratory operation usually adds to the difficulties in determining the extent of the tumor at the second operation, and it is in these cases that recurrence is most likely to take place.

In the differential diagnosis of malignant tumor of the antrum, one must consider giant cell tumor, osteitis deformans, fibroma, adamantinoma, benign cyst, and osteoma. The roentgenogram is a great aid in determining, before operation, the type of tumor and its extent.

### CASES STUDIED

We are presenting a study of 295 patients with malignant tumor of the upper jaw and antrum, who were operated on prior to January 1, 1929, in order to determine the curability of these tumors. There were 141 tumors of the antrum, of which 91 apparently were primary and 50 apparently secondary, and 154 tumors of the upper jaw. The division between primary and secondary tumors of the antrum is not absolute, since difficulty arose at times in determining whether the tumor originated in the jaw or in the antrum; the division is important, however, because of the difference in prognosis in the two groups.

Of the ninety-one primary malignant tumors of the antrum, sixty-three were squamous-cell epitheliomas; six, adenocarcinomas; seven, round cell sarcomas; nine, fibrosarcomas or osteosarcomas; three, lymphosarcomas; two, malignant tumors of undetermined cell-type; and one was a myxosarcoma. The tumor was situated on the right side in forty-two cases and on the left side in forty-nine cases, 46.2 and 53.8 percent, respectively.

Of the fifty secondary malignant tumors of the antrum, thirty were squamous-cell epitheliomas; five, adenocarcinomas; eleven, malignant tumors of undetermined cell-type; two, fibrosarcomas; one was a round cell sarcoma; and one a myxosarcoma. The tumor was situated on the right side in twenty-five cases and on the left side in twenty-five cases.

*Distribution by Age and Sex.*—Of the ninety-one patients in the group with primary malignant tumor of the antrum, forty-eight were men and forty-three were women, an approximately equal distribution between the sexes. The average age of the men in this group was forty-six years and of the women forty-nine years. Of the fifty patients with secondary malignant tumor of the antrum, forty-four were men and only six women, or 88 percent compared to



12 percent. The average age of the men in this group was fifty-three years and of the women thirty-nine years. Of these 141 patients with primary or secondary malignant tumor of the antrum, ninety-two were men and forty-nine women, 65.2 percent compared to 34.8 percent. The average age of the men in both these groups was forty-nine years and of the women forty-seven years.

Malignant tumors of the upper jaw occurred approximately three times as often among the men in this group as among the women, 74 percent, compared to 26 percent. Of the 154 patients in the group, the average age of the men was fifty-four years and of the women forty-four years.

Of the 295 patients in the series, 206 were men and eighty-nine women, 69.8 percent compared to 30.2 percent. The average age of men in the series was fifty-two years and of women, forty-six years.

*Treatment.*—The selection of cases for operation is based on the situation and extent of the growth, the type of tumor, and the presence or absence of metastasis and, in addition, the patient's age, general condition, and ability to return for observation. These patients should be kept carefully in touch with after operation, so that if the growth recurs they can be treated immediately. Patients with extensive primary antral tumors of highly malignant type have a better chance of recovery than those with tumors of the same size but of a low grade of malignancy, so that patients with very extensive, squamous-cell epitheliomas, graded 4, or with sarcomas, are treated.

The treatment of tumors of this type has been outlined by us in previous reports. In most of our cases anesthesia is by intratracheal administration of nitrous oxid, the tumor, as a rule, being approached through the mouth above the alveolar process or through the palate, depending on its situation. Biopsy is made and a fresh frozen section is examined microscopically to determine the type of growth. In addition, lateral rhinotomy may be employed; an incision is made lateral to and below the nose, a portion of the nasal bone removed, and the growth destroyed through this approach. The aim, in treating lesions of low grade is to destroy the tumor completely with diathermy, and with the more highly malignant tumors to establish thorough drainage by means of destruction by diathermy; radium is then inserted where it is required. A protected endotherm point, with the spark gap almost closed, is used in approaching the more dangerous regions about the orbit or the ethmoid bone, whereas a large point, with the spark gap well open, is employed in regions wherein wide destruction will not be of any serious consequence. Preliminary ligation of the external carotid artery is not done as a

routine measure, and is employed only in an occasional case in which secondary bleeding necessitates its use.

Postoperative observation every month or six weeks is advisable in cases of malignant tumor of the antrum. Sequestra are removed as soon as they tend to loosen. A vulcanite dental plate is used to close the postoperative opening in the upper jaw and is much preferable to a pedicled flap brought in from the neck. It is difficult to get a dental plate with artificial teeth that fits satisfactorily over the flap used to close a large postoperative opening in the jaw. Postoperative perforations in the cheek or nose, which occur in a small group of patients, are best closed by tubed flaps brought up from the neck and thorax. In most cases it is necessary to line the flap with a full thickness skin graft in order to obtain a double layer of epithelium and to get a flat graft. We prefer to raise a flap with the upper end in the supraclavicular region and the lower end extending down over the thorax. After delaying it the distal end is carried to the mastoid region and, at the next stage, the proximal end, in the region of the clavicle, which has been lined with a full thickness skin graft, is brought to the area on the face where it is required. This method eliminates the scarring of the neck that follows the use of a tubed flap in the cervical region, as is usually employed. The character of the skin in the supraclavicular region is similar to that on the face and makes for a very satisfactory cosmetic result.

*Results.*—There was but one death in this series during the postoperative period of convalescence; this patient died of bronchopneumonia during an epidemic of influenza.

The results of Holmgren and Öhngren reveal 38.5 percent five-year cures in fifty-two cases of malignant tumor of the antrum. They were able to trace all of their patients, whereas of 141 patients with primary or secondary malignant tumor of the antrum who were operated on prior to January 1, 1929, we were able to trace 118; of these, fifty-three were well without recurrence, which makes 44.9 percent five-year cures among patients who were traced, or 37.5 percent of the number operated on. Hauser reported fifteen cases, with 13.3 percent five-year cures, and Peyton reported eleven cases; two of Peyton's patients are still living, one for more than five years.

Of patients with primary malignant tumor of the antrum, 40 percent of those who were traced were alive without recurrence five years after operation. Among patients with primary sarcoma of the antrum, 72.2 percent of those who were traced were alive without recurrence five years after operation. The fact that many of the growth were of low-grade malignancy accounts for these results.

Of patients with primary epithelioma of the antrum who were traced and the tumor graded, a higher percentage of those with tumors of low grade (grades 1 and 2) were alive without recurrence five years after operation than were those with tumors of high grade (grades 3 and 4), 55.6 percent as compared to 34.5 percent. Of patients with primary adenocarcinoma of the antrum, 33.3 percent of those traced were alive and without recurrence five years after operation; although these tumors are of low grade of malignancy, they are not radiosensitive, and complete destruction with diathermy offers the best chance of treatment. Of patients with secondary malignant tumors of the antrum, 53.4 percent of those traced were alive without recurrence five years after operation. These tumors originate in the upper jaw, and the prognosis is better than with primary malignant tumors of the antrum. Of patients with adenocarcinoma originating in the upper jaw and involving the antrum secondarily, 60.2 percent of those traced were alive and without recurrence five years after operation. Of the entire group of patients with primary or secondary malignant tumor of the antrum, 44.9 percent of those traced were alive and free from recurrence five years or more after operation.

Of patients with malignant tumor of the upper jaw including the palate, 62.7 percent of those traced were alive and free of recurrence five years or more after operation. Of patients with malignant tumor of the upper jaw without involvement of lymph nodes, 68.3 percent of those traced were alive and free from recurrence five years or more after operation, whereas of those with involvement of lymph nodes only 26.7 percent of those traced were alive without recurrence five years or more after operation.

Of the entire group of 295 patients with malignant tumor of the upper jaw and antrum, 236 were traced, and of these 127, or 52.8 percent, were alive without recurrence five years or more after operation.

#### CONCLUSIONS

1. Malignant tumors of the upper jaw and of the antrum are curable.
2. Surgical diathermy and irradiation are the most efficient measures of treatment.
3. Biopsy should be made in all cases at the time of operation as an aid in determining treatment.

TABLE 1.—SURVIVAL AFTER OPERATION FOR VARIOUS CONDITIONS

<i>Condition</i>	<i>Underwent operation</i>			<i>Lived after operation</i>		
	<i>Prior to</i>	<i>Patients</i>	<i>Traced</i>	<i>Patients</i>	<i>Percent of traced patients</i>	<i>Years</i>
<b>Antrum</b>						
Primary malignant tumors	1-1-29	91	75	30	40.0	5 or more
	1-1-24	58	49	12	24.5	10 or more
	1-1-19	21	17	4	23.5	15 or more
Secondary malignant tumors	1-1-29	50	43	23	53.4	5 or more
	1-1-24	42	35	12	34.3	10 or more
	1-1-19	16	12	3	25.0	15 or more
Primary and secondary malignant tumors (total of the two foregoing divisions)	1-1-29	141	118	53	44.9	5 or more
	1-1-24	100	84	24	28.6	10 or more
	1-1-19	37	29	7	24.1	15 or more
Including involvement of palate	1-1-29	154	118	74	62.7	5 or more
	1-1-24	98	66	28	42.4	10 or more
	1-1-19	66	40	13	32.5	15 or more
<b>Malignant tumors of upper jaw</b>						
Without involvement of lymph nodes	1-1-29	136	104	71	68.3	5 or more
	1-1-24	84	55	27	49.1	10 or more
	1-1-19	56	32	13	40.6	15 or more
With involvement of lymph nodes	1-1-29	18	15	4	26.7	5 or more
	1-1-24	14	12	2	16.7	10 or more
	1-1-19	10	9	1	11.1	15 or more
Primary malignant tumors of upper jaw and secondary malignant tumors of the antrum	1-1-29	204	161	97	60.2	5 or more
	1-1-24	140	101	40	39.6	10 or more
	1-1-19	82	52	16	30.8	15 or more
Primary and secondary malignant tumors of upper jaw and antrum	1-1-29	295	236	127	52.8	5 or more
	1-1-24	198	150	52	34.7	10 or more
	1-1-19	103	69	20	29.0	15 or more



# THE PHYSIODYNAMICS OF INTRAVASCULAR FLUID INJECTIONS FOR ROENTGENOGRAPHIC STUDIES

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The chief clinical problem, which I wish to discuss, is a method of visualizing, with the x-ray, the intracranial venous sinuses by the injection of an inert opaque medium.

About four years ago, while carrying out some experimental studies upon vascular repair in the dog,<sup>1</sup> I resorted to the x-ray, in order to study more completely the vascular healing processes without disturbing the wound. As these surgical researches were concerned primarily with studies on sigmoid sinus thrombosis, the visualization of the injured vessel by x-ray promptly suggested the use of an opaque medium and the x-ray as an aid in the diagnosis of sigmoid sinus thrombosis.

Every otologist has at times been confronted with the perplexing problem, not only of determining whether the sigmoid sinus is thrombosed, but, when both ears are infected, which sinus is involved.<sup>1a</sup>

Trendelenburg<sup>1b</sup> was probably the first to suggest vasographic studies when, in 1902, he observed, by x-ray, a moving bullet within the heart chambers. Reithus,<sup>2</sup> Franck and Alwens,<sup>3</sup> and Scheppelmann<sup>4</sup> made intravenous injections of various types of opaque medium in animals, which they studied under the x-ray. They failed, however, to develop a satisfactory medium for human use. In 1920 sodium iodid was used by Berberich and Hirsch,<sup>5</sup> Brooks<sup>6</sup> and Santos.<sup>7</sup> The latter was one of the first who attempted to visualize the vascular lesions in the brain by the injection of sodium iodid directly into the carotid artery.

Sodium iodid produces a beautiful roentgenographic shadow, but when used in sufficient concentrations to produce this shadow it is so severe a vascular irritant that great harm results. I was immediately impressed with this danger when all of my experimental animals died within thirty-six hours after these injections. Even when only five cubic centimeters of a 50 percent solution of sodium iodid was injected into the sublingual vein of the dog such a marked vascular irritation occurred in the lungs that the dog promptly developed an extensive pneumonia with pulmonary edema and pleural effusion.

Obviously this medium is unsatisfactory for human use. Finally, in 1930, Radt<sup>8</sup> introduced a preparation called thorotrast,<sup>9</sup> which is a solution of thorium dioxid with an added colloidal substance which he used in the visualization of the liver and spleen. Allen and Camp<sup>10</sup> have recently reported successful arteriographic studies with this medium. As it is stored indefinitely within the reticulo-endothelial system of the various organs, its eventual harmful effects are undetermined. That it is not without some dangerous toxic reactions is shown by the fact that it has not yet been approved by the Council of Pharmacy and Chemistry of the American Medical Association.

About this time a nontoxic, inert iodine compound was sent to me for experimental studies. This substance had not yet been placed on the market, and was devised solely for intravenous injections for urographic studies. In certain amounts and concentration, it has been proven to be harmless and is quickly and completely excreted by the kidneys. For these vascular studies is fulfilled all of the necessary requirements except one: it did not show a complete shadow of the injected vessel. A faint line along the center of the channel was all that could be identified. This failure was due to the low viscosity of the solution, the center flow and rapid dilution within the blood stream, which will later be explained. Although it contained 52 percent of inert iodine, it did not produce enough of the contrasting shadow. The animal was in no way disturbed by the intravenous injection of the solution, even in large doses, given at frequent intervals, but it became apparent that other factors played a part in the successful technic of the procedure. Certain factors were conceived and have been proven as necessary for satisfactory visualization and use in the human, and it is along the lines of physiodynamics and chemistry that certain phases of the problem are now being studied. The factors which play such an important part are as follows:

1. The opacity of the solution.
2. Complete filling of the channel that is to be studied with this opaque solution.
3. Limited miscibility with the blood in the region of investigation.
4. Immediate dissipation by gradual miscibility and complete elimination at some more distant point.
5. A viscosity which will permit ease of injection, yet prevent rapid dilution by the blood.
6. A solution which will itself remain true to its purpose without chemical change, deterioration or otherwise becoming useless by the process of sterilization. The solution must be nonirritating to

both the vascular endothelium and surrounding tissues, produce no harmful pathologic changes when mixed with blood, and should be harmless to all organs and free of all functional disturbances.

For injecting the sigmoid sinus, the sinus should be exposed in the usual manner following the mastoidectomy, and the solution injected directly into the blood stream, the needle being inserted distal to the site of the suspected lesion. The needle should be large enough to permit the chilled solution of glucose and opaque medium to enter rapidly but not so large as to make an unnecessary tear in the sigmoid sinus wall. The syringe should be equipped with finger grasps and the needle either screwed or locked in place in order to avoid obvious accidents. The bevel of the needle should not be too long, and in some instances the needle should be bent at an angle of 30 degrees, so that the edges of the mastoid wound do not interfere. The x-ray picture must be taken while the solution is flowing in and just prior to the last one or two cubic centimeters of injection. Care should be taken that none of this solution escapes into the mastoid wound because of the resulting confusing shadows. As the examined sigmoid is farther from the plate, any light angulation may produce a confusing shadow (Fig. 1), so that interpretations should be made accordingly. Stereoscopic films make it much easier to differentiate the vascular shadows and should be used when possible. Care should also be taken to keep the hands of assistants and the operator out of the field, as finger shadows are easily confused with vascular shadows. Better pictures are secured with a patient under anesthesia while the mastoidectomy is being performed, but, with a cooperative patient, they may be taken without any anesthesia whatsoever, as there is no pain connected with the procedure.

I believe that the use of this contrast medium for the differential diagnosis as well as the definite diagnosis of the intravascular lesion within the sigmoid sinus, offers our first positive means of determining just what has taken place within this vessel and establishes a definite method of procedure based upon more accurate preoperative evidence. Frenckner<sup>11</sup> has reported some experimental studies, which have recently been published. His work was carried on independently of mine, and, as he states, he has not used his method upon the living human. He also used thorotrast, which has the disadvantages previously mentioned.

I am now working with a chemist in the development of this opaque medium and I find that there are about thirty-five ways in which this chemically inert substance may be compounded. The development of a particular application of this medium has come

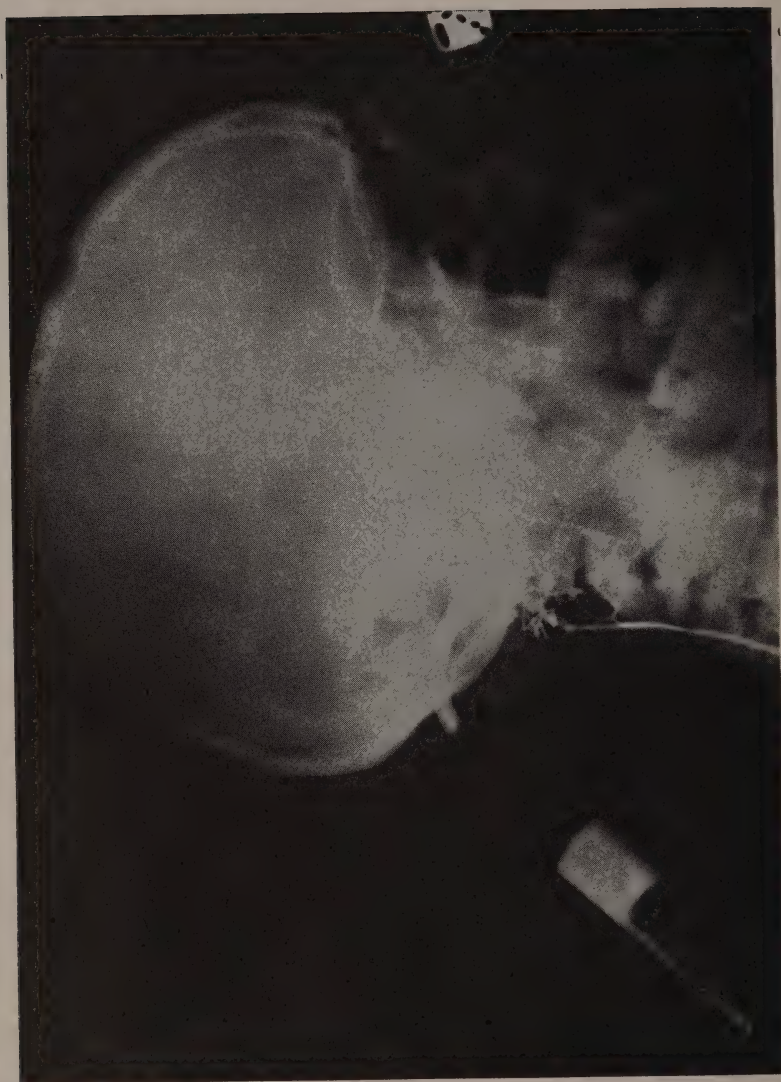


FIG. 1. Post-mortem injection of opaque medium directly into the opened sigmoid sinus. The patent internal jugular vein is clearly shown filled with opaque medium. It appears to be misplaced because of the malposition of the head.



about by combining it with glucose and then chilling. Various factors still remain to be worked out, and while I feel certain that the method that I have now used is safe, yet there remain some objectionable features which I hope to soon have removed and will report in a separate paper.

#### THE COMPOUND AND ITS PROPERTIES

The solution which I have prepared and found to be the most satisfactory is a mixture of glucose and inert iodine compound of sufficient opacity and viscosity to obviate the deficiencies of solutions previously used.

This solution contains approximately 19.2 grams of iodine per 100 cubic centimeters as combined iodine. This iodine is in chemical combination with carbon, hydrogen, oxygen and sulphur, in the form of a sodium salt. This iodine compound with 71.5 grams of glucose in water solution, sufficient to make a total of 100 cubic centimeters, constitutes a solution of proper viscosity and opacity when used at a temperature sufficiently below body temperature to give intravascular viscous flow.

The purpose of viscous flow is to prevent rapid dilution of the opaque medium within the blood stream. Obviously the greater the concentration of the opaque medium near the obstructive lesion the more detailed will be the x-ray shadow.

The choice of a chilled and more viscous medium is unique in its application in that it contradicts the former practice of using a warm and less viscous solution. The former practice was carried on under the misapprehension that fluids injected into the blood stream at a temperature lower than that of the body were injurious. My former erroneous idea was that a less viscous solution was necessary in order to permit a rapid injection and thorough mixing of the opaque medium with the blood at the site of inspection.

Chilling of the solution was determined quite by accident. It was necessary to haul these animals a mile from our experimental laboratory to the x-ray room where these studies were made. In order to conserve time, my assistant filled all the syringes separately with the opaque solution and placed them in an open car, while we anesthetized the dogs with amytal and morphine, preparatory to the x-ray procedure. The solution, which was in the syringe, was in the open air for about one hour, and must have been only a few degrees above freezing, and at the time of the injection was probably between 40 and 50 degrees F. Although accurate temperatures of the injected solutions were not made at this time, the Weather Bureau

has since sent me a report made on that date, November 10, 1932. The maximum temperature was 36 degrees F. and the minimum temperature was 28 degrees F. When the intravenous injections were started in the usual manner this accidentally chilled solution had become so viscous that it was almost impossible to inject it into the sublingual vein. In fact it went in so slowly, and we were so certain of poor results, that we did not develop our films that evening. It was a very pleasant surprise, the next morning, to find that these were the first good films showing complete intravascular details with this inert and harmless type of solution. At once it was apparent that the lowering of the temperature with increasing viscosity was the important factor in the success of the procedure.

By referring to the viscosity temperature table for this solution, the optimum temperature for each desired use may be used.

SOLUTION VISCOSITY TEMPERATURE TABLE

No. 1			No. 2					
Temperature	20 Percent by Weight	40 Percent by Weight	Wt. %	25°	30°	35°	40°	50°
0° C	38.18	148.2	34.94	33.3	28.8	24.7	21.6	16.9
10° C	26.62	98.30	42.33	52.4	44.0	37.4	32.2	24.3
20° C	19.67	62.23	49.33	88.2	72.2	60.2	50.9	36.9
30° C	15.10	43.93						
40° C	11.97	32.61						

It is a well-known scientific fact that any variation in temperature produces a corresponding variation in viscosity of any solution or liquid. The importance of viscosity was not fully appreciated, and although glucose was used in the attempt to fulfill this requirement, yet it was not until the glucose was chilled by accident that a proper viscosity was obtained, thus proving that the temperature of the solution plays a very important part as a factor in obtaining good x-ray results.

#### PHYSIODYNAMICS OF INTRAVENOUS INJECTIONS

Although the intravascular injection of foreign substances is a very old procedure, there are still certain technical factors which are not thoroughly understood. It has finally remained for the physicist to develop certain accurate mathematical laws governing the flow of fluids. Obviously the physiodynamics of this problem, which we are now discussing, becomes a very important factor in the proper

application and the correct interpretation of these intravascular studies, which are to be made by the use of this intravascular medium.

I have been fortunate to have the assistance of Dr. Robt. M. Isenberger, professor of pharmacology, University of Kansas School of Medicine, and Mr. E. R. Harrouff, Kansas City, whose training in chemical engineering and physiological chemistry has especially adapted him to the interpretation of some of these studies. Before entering into the highly technical phases of physiodynamics it may be well to describe just what is desired by the injection of this opaque medium into the blood stream. Granted that the previously mentioned objectionable factors have been removed the complete accuracy of the studies of the suspected intravascular lesion is dependent upon the opaque medium completely filling the vessel throughout its course, so that any narrowing or deformity from its normal contour can be plainly seen in the x-ray shadow.

In a more detailed scientific study of the problem we are at once confronted with straight line and turbulent flow, and the velocity at the point of change of these two flows which is called the critical velocity. The equation of Poiseville's law, for straight line flow, and Fanning's equation for turbulent flow, are used for mathematical precision in fixed tubes. The full proof of the facts found by discovery, as outlined up to this point, lies in the field of pure science. There are definite laws expressed by algebraic equation governing the flow of fluids in tubes. The matter of fluid flow was first investigated by Poiseville,<sup>13</sup> a French physician, in 1843, and later a law was formulated bearing his name. Poiseville's law of fluid flow has been much refined in the light of modern knowledge, so today we have a definite formula for fluid flow, wherein the diameter of the tube, the length of the tube, the quantity or the volume of flow, ( $y$  equals  $\pi r^4 t / 8 L Q$ ), wherein the viscosity of the flow is expressed in definite units of force called poise and centipoise, in the metric system.\*

The application of Poiseville's law to this problem involves also the equation of straight line flow and Fanning's equation for turbulent flow. The equation is ( $dp$  equals  $4 f \text{ flpu}^2 / 2 \text{ gd}$ ). The equation for straight line flow is ( $dp$  equals  $32 \text{ ulu} / \text{gd}^2$ ).<sup>\*14</sup>

The underlying facts and theory to be correlated in this problem is whether a warm or cold solution gives better results when injected into the blood stream, which has a definite viscosity, temperature, volume and rate of flow in any given blood vessel of known size and location. The location is only incidental, in that it indicates

\*  $y$  equals Greek Mu.  $p$  equals Greek Rho.

the diameter of the vessel for injection, being more or less as experience has shown to be required. Other factors are the flexibility of the tube walls, pulsations due to heart action and muscle activity, bends in the tube, etc. The pulsations are damped out by the elastic walls of blood vessels. When all of these factors have been settled by experiment, then it may be possible to make an injection into the blood, with some degree of certainty as to the outcome.

The physical and chemical factors of the blood have long since been definitely determined and found to be constant for all practical purposes; our attention becomes centered on what the physical and chemical constants of any fluid must be for successful x-ray pictures, after injection into the blood vessel and flowing blood stream having these fixed physical and chemical constants.

The evident temperature effect requires explanation in such manner as to correlate the facts with the underlying theory. The equations of straight line and turbulent flow make apparent the relationship of the different physical factors involved in the flow of any one liquid at any one temperature and the point where straight line flow changes to turbulent flow. The laws of flow show that the greater the viscosity of any liquid the more it tends to straight line flow and *vice versa*. Straight line flow is understood to mean flow without mixing or rolling in eddy currents such as occurs in turbulent flow.

Since liquid of low viscosity—that is, very free flowing—tends to turbulent flow, any such solution soon spreads itself through a large volume of blood, in this case resulting in such extreme dilution of the opaque medium as will prevent obtaining good x-ray pictures.

The problem becomes more complicated by the fact that two liquid solutions are involved and at two different temperatures. It is evident that to obtain good x-ray pictures straight line flow is required, or in other words more viscous flow, for the reason that extreme dilution of the iodine compound must be prevented up to the point of the obstructive lesion.

The rate of flow for this problem has not been definitely established, but it is a factor in knowing the time required for the opaque medium to reach a certain distance from the point of injection, depending upon the cross sectional area of the blood vessel. The theoretical equation for rate of discharge through an orifice is  $V$  equals  $C\sqrt{2gh}$ . It is well known that a lowering of temperature increases viscosity, and we know that viscosity of the blood remains practically unchanged. We know from the flow laws and formula that any liquid less viscous than the blood will cause turbulent flow,



which means rapid mixing of the solutions, consequently more rapid dilution of the solution. The opposite must be the objective desired. Temperature is found to play the more important part in the mixing of two solutions of different viscosity.

One of these solutions, the blood, we know remains at a constant temperature and viscosity, and any solution injected will eventually become the same temperature, followed by more intimate mixing and dilution of the iodine compound. This is desired at a point away from the region of inspection but not at the point of inspection. Since a solution of low temperature accomplishes these results the next answer to be sought is, "Why?"

This may best be illustrated by the example of two liquids, say syrup and water, each having different viscosities, as we know. Assume the water is at a fixed temperature of 98° F., at which temperature it has a viscosity coefficient of .690 centipoises. Then assume the syrup of 40 percent sugar content also to be at 98° F. temperature and to have a viscosity coefficient of approximately 35 centipoises. The two solutions are now mixed and the time rate of complete mixing observed, which is then repeated with the same solution, except for the syrup which has been previously cooled to, say, 40° F. It will be noted that the time required for complete miscibility to occur is much longer.

With these same solutions and physical conditions we next observe what happens in flowing through tubes. We find that the solution of syrup at 40° F. flows more slowly and without turbulence. If followed by water or blood, the center flows faster and the blood takes this center and smaller channel, leaving a layer of more viscous liquid next to the tube wall to be gradually washed along.

However, in the case of injection into the blood vessel a different condition exists. The blood vessels are warm and will soften the layer next to the wall, thus reducing the viscosity in this thin layer, forming a cylinder of lubrication next to the vessel wall, causing the middle stream of more viscous solutions to be pushed ahead as a dense plug by the blood, just as a lubricated piston moves along in a cylinder, and although the method of lubrication is different the effect is the same. This keeps the blood vessel fully distended and also filled completely with opaque solution. It is readily seen that a peripheral layer of lower viscosity of the injected solution occurs after injection, due to the warm blood vessel walls increasing the temperature in the peripheral layer of injected fluid.

The moving plug persists through the entire region of inspection, but is later churned up with the blood in the heart and elimi-

nated from the body, with no harmful effects to any organs or tissues, nor any functional disturbances to any of the organs or tissues.

The smaller the blood vessel the less viscous the injected solution needs to be, because there is little or no turbulent flow in small tubes. This fact makes possible the use of a warmer solution than is injected into larger vessels, and consequently a smaller needle can be used.

A further detailed study is necessary, of the equation of flow and the point of critical velocity, which is the velocity of flow where turbulent motion begins. The critical point of flow is obtained by equating the equations for straight line flow and turbulent flow for equal pressure drop. It may be assumed that the pressure drop is the same in the blood stream at any one point, therefore it is proper to equate these two equations ( $dp$  equals  $32 \gamma \text{ lu/gd}^2$ ), and ( $dp$  equals  $\gamma f \text{ lu}^2/2gd$ ).

Therefore equating the Poiseville and Fanning equation, we have ( $32 \gamma \text{ lu/gd}^2$  equals  $2f \rho u^2 2gd$ ), and from this ( $U$ ) equals  $942 \gamma/p d$ ) for all fluids for values in feet and decimal feet.

In this problem the object sought is a greater critical velocity for the injected fluid than that of the blood. The extent of this difference is determined by experiment where the values are selected that produce the best results consistently. The use of the laws and equations of flowing fluids make possible a positive scientific analysis of what is taking place when it is desired to obtain a definite result from intravascular injections for x-ray pictures.

From such experiments data are obtained for finding the definite physical constants of the solution to be injected, which experiment has proven to give the best result.

The entire process can be studied with blood and solution in the laboratory in glass tubes in a glass water bath adjusted to body temperature. The flow of blood can be simulated in the tubes by actually using blood itself. The tubes are so provided that injection of the opaque fluid or solution can be made similar to a blood vessel injection.

Experiments in rubber tubing can be carried out also. These experiments are now being conducted and will be reported in a separate paper. Withdrawal of blood into a syringe, filled with 50 percent glucose, prior to intravenous injection, shows the relative viscosity and miscibility.

Diffusion plays no part of consequence in the problem of intravascular injection because it is far too slow a process to effect dilution of the opaque medium to any appreciable amount.

The energy and time required to cause diffusion are too large to be a factor of dilution in the time devoted to making x-ray pictures as has been described.

The study of injections into the fixed tissues, such as muscles, brain cavity and other organs, is in progress. Such injections appear practical. The lymph stream is the vehicle that carries the solution to the desired location in the tissues. This is a problem to be dealt with mathematically in the field of infiltration or convection.

### CLINICAL APPLICATION

The use of this medium for intravascular studies has rather unlimited possibilities.

From previous description of the manner of the physiodynamics of the problem we are dependent on the circulating blood to transport the solution; therefore it must always be injected distal to the point of the suspected vascular lesion. In the present stage of development it is not so well adapted to arterial studies as to venous studies, but fortunately our clinical problems are more often confined to the venous system. It is distinctly more applicable for the study of lesions within the venous system than in the arterial.

For the study of intracranial venous lesions the principal channels that are involved are those lying adjacent to the cranial vault and readily accessible to direct injection. The ideal medium would be one that could be injected into the arterial supply, *i.e.*, the carotid artery, carried to the brain, and the shadows studied upon their return through the venous system. Obviously this calls for a large amount of solution, which on account of the overlapping of the vascular shadows may produce a confusing picture. This has already been tried with only fair success. In the dog which has a much smaller brain in comparison with the human, injections into the carotid artery have been made. The larger arterial vessels can be identified as well as the returning venous flow, yet the medium is so diluted on its return that the contrasts are not always satisfactory. (Figs. 2a and 2b.)

It may be desirable to study all of the intracranial dural sinuses, by one injection, as illustrated by the following case:

E. H., a forty-year-old man, on October 22, 1934, was admitted to the hospital with a large carbuncle on the back of the neck, which after five days was radically removed. Two days following this operation he had a slight chill and convulsion and immediately following he collapsed into unconsciousness from which he did not recover. He developed a flaccid paralysis of his right arm; his temperature varied from 101 to 102.5° F., his pulse rate was in



FIG. 2a. This picture shows the shadow, in the right internal jugular vein, of the returning solution that was being injected into the right carotid artery. The external jugular vein had previously been obstructed. The brain shows a distinct opacity, but no vascular detail can be made out.

FIG. 2b. This shows the same animal with the picture taken six minutes later. The brain cavity is clear and there are no remaining shadows in the veins.

the 80s, his respiration in the low 20s, with a leucocyte count of 16,400, and his spinal fluid was normal with an increase in pressure.

Dr. Frank Teachenor of Kansas City, who had called me to make these radiographic studies, made a buttonhole trephine over the longitudinal sinus near the junction of the frontal temporal bone. Through this opening I injected the radiopaque solution. In the first series concentration was not great enough to give a distinct shadow, but later, by chilling and increasing the viscosity and the



concentration of the solution, very satisfactory shadows were procured, which definitely ruled out any blocking in the intracranial sinuses, as the shadow could be followed from the point of injection down to the edge of the plate into the internal jugular vein. (Fig. 3.) An accident occurred during one of these injections (Fig. 4) which emphasized the value of a nonirritating and quickly absorbable solution. The point of the needle pierced the floor of the exposed longitudinal sinus, which permitted the opaque solution to leak out be-

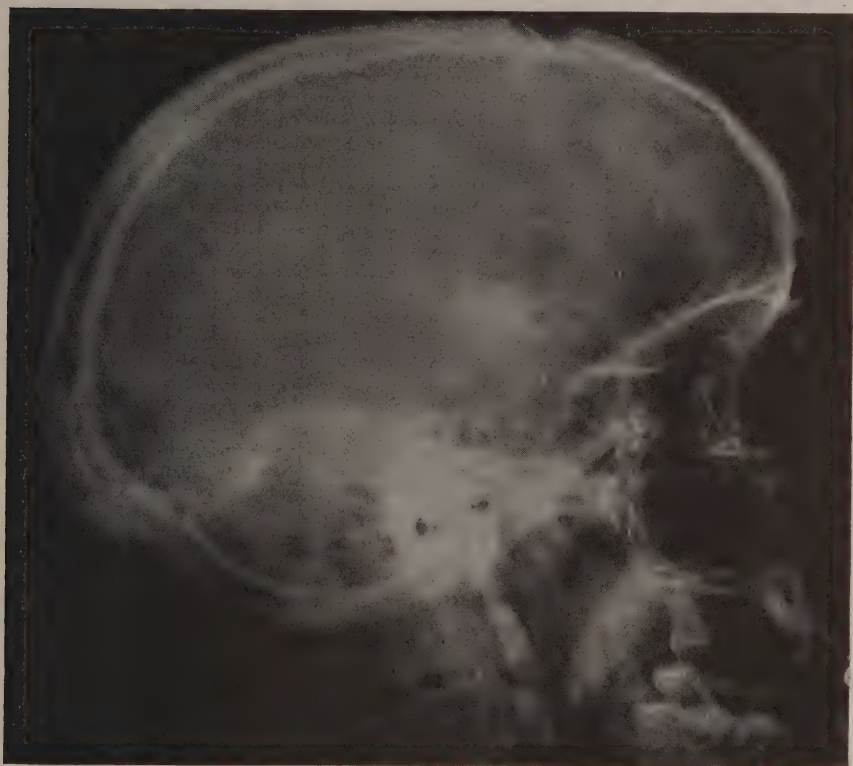


FIG. 3. A complete shadow of the longitudinal sinus from the point of injection at the site of decompression, including the jugular bulb and the internal jugular vein. This demonstrates that the vessel is patent throughout.

tween the two hemispheres of the brain. There was no reaction by the patient and the accident was discovered only when the film was developed. As soon as additional films could be taken, which was about twelve minutes later, this shadow had completely disappeared. Finally, after this man had died of an extensive encephalomalacia of the left frontal lobe, which would have eventually developed into a brain abscess, could he have survived long enough, injections of the

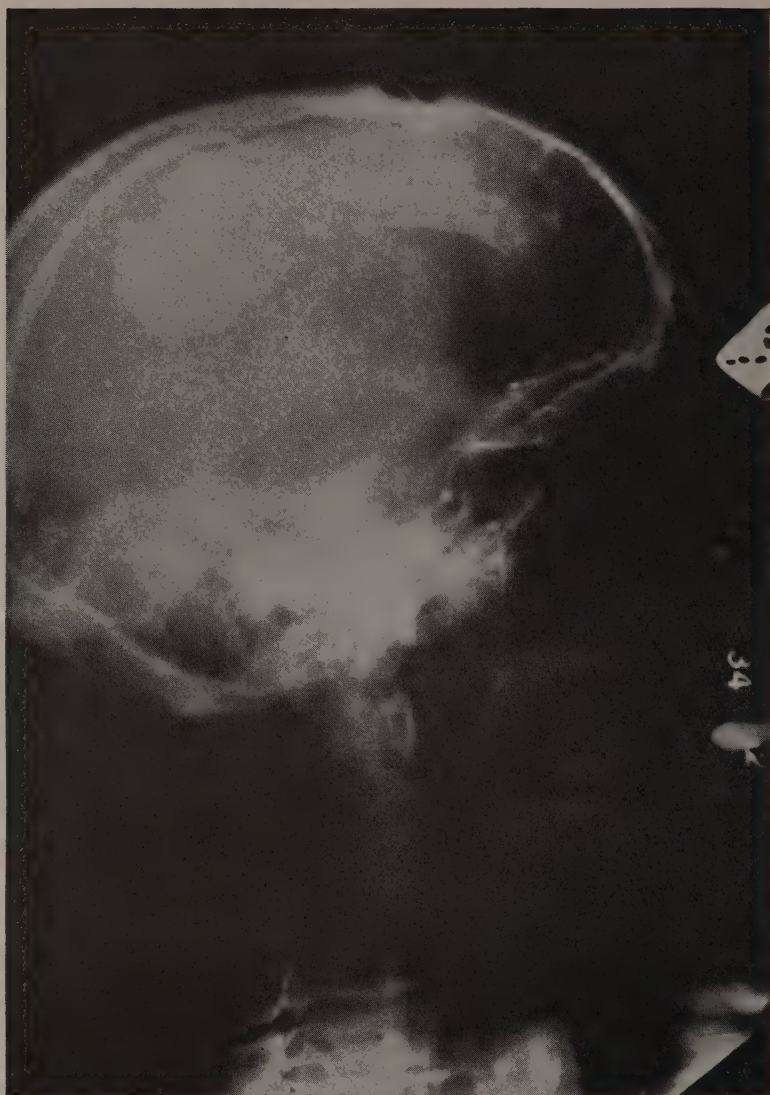


FIG. 4. Shadow produced by the accidental injection of this opaque solution through an opening made by the point of the needle in the floor of the longitudinal sinus at the site of injection. As soon as another picture could be taken, which was about ten minutes after the injection, this shadow had completely disappeared.

opaque solution were made into each sigmoid sinus, which brought out certain points in the x-ray technic.

One distortion (Fig. 1), which apparently shows the internal jugular vein going down the back of the neck, was caused by the improper position of the head and neck at the time the picture was



FIG. 5. Shadow produced by the accidental escape of the opaque medium beneath the dura, while injecting the sigmoid sinus. This in no way disturbed the patient and was quickly absorbed.

made. Another picture illustrated the possibility of using this medium in the postmortem studies of the anatomy of certain vascular areas. The thorax had not been opened, and the lower end of the jugular vein was still blocked by postmortem clots when this solution was injected under moderate pressure. There was a retrograde extension into the tributaries, which apparently extended into the pterygoid and pharyngeal plexus. While this is principally of academic interest, it suggests the possibility of using this or some other radiopaque medium in postmortem studies, where necropsy permits



cannot be obtained or where it is undesirable to disturb certain vascular areas. It may be also used as a matter of permanent record to illustrate the vascular lesion.

When the suspected lesion is quite distant to the point of injection it is obviously necessary to use a sufficient quantity of the solution that would carry beyond the site of suspected lesion, otherwise confusion may result as to the interpretation of the termination of the shadow, as it could not be determined whether this shadow was due to actual blocking of the vessel or the normal limits of the intravenous opaque medium. This again emphasizes the value of glucose as a vehicle. In the dog, I have been able to determine accurately, lesions along the course of the inferior vena cava by the injection of this medium into the saphenous vein. While I have not yet had the opportunity to make the same observation in the human, I see no reason to believe that it would not be quite satisfactory. As it is nonirritating, inert and readily absorbed, it should also be an ideal medium for the study of suspected spinal cord lesions. I have used it in the study of sinus tracts. Because it is absorbed so readily by the large surface of granulation tissue, the x-ray picture should be made immediately after the injection. Again, it has this advantage, that we need not be concerned about any local irritation or the retention of any foreign material which might be confusing when additional x-rays are made.

For the diagnosis, localization and accurate determination of the boundaries of other vascular lesions, such as arteriovenous aneurysms, it is distinctly applicable. It should be of distinct service in the differential diagnosis of thrombophlebitis of the extremities. Not only are its inert properties advantageous, but it can be injected remotely away from the site of the acute inflammatory reaction. I propose to use it also in the differential diagnosis of selected cases of retrobulbar abscesses and cavernous sinus thrombosis, keeping in mind the danger of traumatizing the angular vein.

The following cases illustrate some of the clinical applications of this problem:

#### REPORT OF CASES

*Case 1.*—D. R., a nine-year-old boy, on June 12, 1934, four days following a bilateral mastoidectomy, with the exposure of both sinuses, had a hard chill followed by a sharp rise in temperature. His blood culture was positive. A swollen right elbow indicated a metastatic involvement. Each sigmoid sinus was injected with the solution, and the obstructive lesion was found by x-ray to be in the vessel, which had the smooth normal appearing wall.

While the last injection was being made, a sudden warning to avoid the uncovered overhead wires, caused me to accidentally pass the point of the needle



through the floor of the sinus and inject some of the solution beneath the dura. (Fig. 5.) The entire procedure was done without any anesthesia and the patient showed no reaction to this accident and has completely recovered from the injection. The x-ray pictures taken soon after the accident failed to reveal any evidence of a remaining shadow. This accident illustrates the necessity of using a solution, which possesses all possible safety factors.

*Case 2.*—S. F., a six-year-old girl, for whom I had removed an infected thrombus from the sigmoid sinus in 1932, and repaired with a viable muscle flap, was selected for use of this opaque solution, to prove that the sigmoid sinus had recanalized. Without any anesthetic, this child was placed on the x-ray table, and a lateral x-ray was made of the head and neck for a control. This opaque solution was then drawn into a five c.c. glass syringe, and with a 26 gauge needle, two c.c. were injected through the skin into the sigmoid sinus. As the concentration of the opaque medium was very low, only a faint shadow was seen, but there was enough solution passed along to definitely establish the patency of the vessel. The patient was not disturbed and immediately left the hospital after this examination.

*Case 3.*—N. T., a five-year-old girl, who was convalescing from measles and had a bilateral suppurative otitis media, suddenly had a hard chill. Her temperature rose to 104°, with a 26,000 leucocyte count. The chill was repeated twenty-four hours later. Under ether anesthesia a mastoidectomy was performed and the sigmoid sinus was exposed. The iodine compound and glucose was injected, but the x-ray showed a blocking near the bulbar end, which also was indicated by the shadow of the solution in the deep posterior occipital vein. Three injections were made on this child, and in no way was she disturbed by the procedure.

*Case 4.*—R. T., a fourteen-year-old boy, was admitted to the hospital, March 24, 1934, in whom for the past year I have had the distressing experience of attempting to establish a better drainage from a brain abscess of cranial osteomyelitic origin. Injections of this opaque media into the fistulous track have established leads, which when followed, opened into new abscess cavities. At no time has the patient been disturbed by these injections, which have been made with only the use of a small amount of gravity pressure. Care should always be taken to avoid the rupture of the pyogenic capsule, enclosing the brain abscesses. If some of the solution escapes upon the scalp, confusing shadows will result. Where a definite abscess is found and pus is aspirated, if the shape, size, and location are desired, the solution may be injected in the amount of one-half of that of the pus, which has been removed.

The rapidity of the absorption of this solution and its prompt disappearance from the site of the injection is well illustrated by the following case:

*Case 5.*—A twenty-six-year-old man with a draining fistula over the anterior surface of the middle third of the leg, of two years' duration, was injected with four c.c. of a warm opaque solution. The solution could be felt beneath the skin as it entered the subcutaneous granular tissues.

The roentgenologist did not know that the injection was being made and was not ready to take the picture until about four or five minutes later. By

that time the solution had so disappeared that only a faint shadow was obtained. The relative opacity of the solution is well illustrated by the droplets that can be obtained within the metal needle on the syringe, and it is well to always show some of the solution outside of the body on the plate, in order to determine the relative opacity.

### SAFETY FACTORS

The opaque media, which I have described for the study of vascular lesions by x-ray seems to possess all the necessary safety factors, as it is inert, nontoxic, does not produce embolic phenomenon, is nonirritating to the vascular endothelium, is quickly and completely eliminated and, even when accidentally or intentionally injected outside of the vascular system, such as the subdural space, is without harmful effects. There is one factor, however, which must be constantly kept in mind—that is, that the patient may have an allergic hypertonic susceptibility to any form of iodine. Proper tests should be made to determine this factor prior to injection. Although this iodine is firmly bound and apparently inert, it might react badly in these allergic patients. Glucose seems to be a safe and proper vehicle, and lowering the temperature of the injected solution from 20 to 30 degrees F. below normal body temperature apparently produces no harmful effects to the patient. Finally, the paramount factor in the use of any therapeutic or diagnostic procedure must always be the safety of the patient.

Dr. Galen Tice, of the University of Kansas School of Medicine, and Mr. Mark Carroll, my technical assistant, have been very generous in their help with this problem.

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# CANCER OF THE TONGUE AND LOWER JAW

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Owing to the propaganda which has been carried on by the medical profession, patients are presenting themselves earlier than they did ten or fifteen years ago with precancerous and malignant lesions about the mouth. It is essential that physicians in general become familiar with the methods of treatment which should be employed in treatment of malignant disease in various situations about the mouth, so that the patient may receive the best treatment. The physician who first sees the patient should assume a personal responsibility in seeing that the patient obtains the most efficient care possible.

## CARCINOMA OF THE TONGUE

Patients frequently consult a physician on account of symptoms which are referable to the tongue, and because of various types of benign conditions which they feel are malignant. One of the most common examples of this group is the patient who has geographic tongue, with multiple plaques of epithelium which change from day to day. Another type of patient is one who has deep fissures in the tongue. These fissures are normal but the patient feels that the condition must be malignant or precancerous. The soreness or enlargement of the small lymph nodes at either side of the base of the tongue frequently produce symptoms that worry this group of patients a great deal. Lingua nigra or black tongue, when present, gives rise to much mental trouble. Submucous thrush, which is characterized by irregular areas and patches about the tongue and cheeks, and by some ulcerated points, also is the cause of much worry. Patients who worry about these conditions practically all have psychoneurosis; they carry a looking glass with them and are constantly looking at their tongues; usually, the minute they are out of bed in the morning, this is the first thing that they do. They need to be assured that these conditions are not malignant and to be told that malignant lesions of the tongue usually appear like an open sore or like a cold sore that will not heal. Many of these patients are women who must be told that carcinoma of the mouth is very rare among women. These patients also worry about the possibility of some type of growth developing at a later period, if one is not present at the time. They should be assured that this does not occur. I feel, however, that any snags of teeth or other sources of irritation should be removed as a precautionary measure.



Certain benign lesions of the tongue, such as lymphangioma which affects children, are readily treated with radiation. Some patients who have huge tongues may obtain complete relief and their tongues may be returned to their mouths, even after this has been impossible for many years. The hemangiomas, too, are cleared up with similar treatment, in cases in which they affect infants and young children. If the patient is an adult treatment by means of a protected diathermy point gives better results than does radiation.

The differential diagnosis of malignant disease of the tongue must include actinomycosis. In the latter condition there is a hard mass in the tongue. This mass may be only 0.25 cm. in diameter or it may be large enough to involve half of the tongue. A wide excision of the small lesions and thorough drainage and packing of the larger lesions, along with radiation and the administration of large doses of potassium iodide should control the condition. The actinomyces are readily demonstrated as little yellow granules, which can be picked up on a knife, and after being washed on a slide, can be diagnosed readily by their club-shaped appearance. Actinomycosis of the mouth is usually caused by a small foreign body of some type, which makes a nidus through the mucous membrane. Actinomyces then develop on the distal end of this foreign body. I have obtained several such specimens, which demonstrate the method of development of this disease in the mouth.

Blastomycosis also may affect the tongue, and may present the appearance of a low-grade carcinoma. A specimen at biopsy sometimes will simulate tuberculosis with giant cells, and unless the pathologist shades down the field so that he can see the refractile bodies the lesion sometimes is thought to be tuberculous. Tuberculosis of the tongue may occur as a primary or a secondary lesion. It may occur as a small tuberculous nodule or as a lupus. The secondary tuberculous lesions which are frequently present in the tongue are secondary to pulmonary tuberculosis. They present a ragged dirty ulcer. The treatment depends on the general tuberculous process. If this is under control, the destruction of the lesion with surgical diathermy allows of healing.

Syphilis of the tongue may be present as a primary lesion, a secondary lesion, or a gumma. The relation of syphilis to carcinoma of the tongue is generally well known. From 15 to 20 percent of carcinomas of the tongue are secondary to syphilitic leukoplakias or deep fissures which frequently occur in the midline of the dorsum of the tongue. Syphilitic leukoplakia frequently is very hard and one may be able to obtain tissue from several parts of the syphilitic tongue which

will show all the stages from the atrophy and leukoplakia to the epithelial hyperplasia and carcinoma. Patients who have syphilitic leukoplakia of the tongue should have these areas widely removed as they are potentially malignant and should be treated as such. The same is true of the thickened leukoplakia of the tongue which is not of syphilitic organ. A wide excision with the cautery, or removal with surgical diathermy, is preferable to any other method. The fact that there are multiple lesions of the tongue does not exclude the possibility of a malignant lesion. I have seen patients who had three distinct areas of carcinoma, that were secondary to a syphilitic condition; each one was a squamous-cell epithelioma, grade 3.

The presence of an old jagged tooth near a malignant ulcer always makes one think that it might be a factor in the production of carcinoma. The etiology of carcinoma, of course, is indeterminate, and any areas of irritation should always be removed. In carcinoma of the tongue, I feel that the best treatment is a wide removal of the growth with diathermy or cutting cautery, if possible, and a bilateral dissection of the submental, submaxillary, and upper cervical lymph nodes on the involved side, if there is no involvement of the lymph nodes. If the lymph nodes are involved, a block dissection on that side should be done. The highly malignant lesions which occur frequently at the base of the tongue, may be treated with radium inserted directly into the tumor, and with radiation used externally. The removal of the lymph nodes of the neck, however, I feel is a very essential part of the treatment of carcinoma of the tongue. I see many at the present time in which the lesion of the tongue has been removed, only to have involvement of the upper cervical lymph nodes appear. This makes the outlook from any form of treatment very questionable. The end results in carcinoma of the tongue are shown in Table I. It shows very well the fact that a patient who has involvement of the lymph nodes has about a quarter the chance of getting well as one who does not have involvement of the lymph nodes.

#### EPITHELIOMA OF THE LOWER JAW

One of the most common neoplasms that is seen in the lower jaw is epulis. An epulis appears as a little red cherry-like tumor along the alveolar process between the teeth. Microscopically, it may be a fibroma, fibrosarcoma or a giant-cell tumor. The important thing, however, is that it usually originates from the periosteum of a tooth socket so that it is essential to remove the adjacent teeth and to destroy the periosteum of the tooth socket at the point where the tumor is attached, in order to prevent recurrence. One must remem-

ber that a very rapidly growing malignant tumor frequently may appear as an epulis-like growth on the alveolar process. From time to time at The Mayo Clinic, we see patients who have had very conservative treatment for what was a highly malignant tumor of the jaw. This has been because a microscopic examination was not made and a careful history was not taken. The rapidly growing tumor must be taken care of in a very thorough manner, while the

TABLE 1.—EPITHELIOMA OF THE TONGUE; FIFTY-EIGHT PATIENTS SURVIVING FIVE OR MORE YEARS AFTER TREATMENT

<i>Lymphatic involvement</i>	<i>Patients operated on</i>	<i>Patients traced</i>	<i>Lived five or more years after operation</i>	
			<i>Number</i>	<i>Percent of patients traced</i>
Nodes not involved but dissected* .....	59	58	29	50.0
Nodes not involved clinically and not dissected† .....	40	36	19	52.8
Nodes involved and dissected* .....	57	56	8	14.3
Extension to floor of mouth.	6	6	2	33.3
Total .....	162	156	58	37.2

\* Cases in which there was extension to the floor of the mouth not included.

† Nodes not dissected because of type of growth, age of patient, and so forth.

epulis is benign. These neoplasms may appear a great deal alike clinically. The epithelioma of the lower jaw may originate in a previous papillary leukoplakia or the first symptoms may be a loosening of a tooth, starting in the body of the jaw. I feel that in these cases thorough destruction of all precancerous lesions with surgical diathermy gives the best chance of getting rid of the trouble permanently. In the lesions that have spread onto the cheek, the removal of the submental and submaxillary lymph nodes adds considerably to the chance of preventing extension to the neck. The end results in epithelioma of the lower jaw are shown in Table 2.

TABLE 2.—MALIGNANT GROWTHS OF LOWER JAW; NINETY PATIENTS SURVIVING FIVE OR MORE YEARS AFTER TREATMENT

<i>Lymphatic involvement</i>	<i>Patients operated on</i>	<i>Patients traced</i>	<i>Lived five or more years after operation</i>	
			<i>Number</i>	<i>Percent of patients traced</i>
Nodes involved .....	45	38	11	28.9
Nodes not involved .....	142	113	79	69.9
Total .....	187	151	90	59.6

## MALIGNANCIES OF THE NASOPHARYNX

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Tumors of the nasopharynx and adjacent anatomic regions are quite varied and as yet offer many problems to the pathologist.

New methods of treatment have stimulated an intense interest in the structure and classification of the tumors of the mouth, tonsils and pharynx, and many changes in nomenclature have occurred in recent years. The relative incidence of the various types of malignancy has been altered as a result of restudy and reclassification.

The grading of tumors and the attempts to estimate the degree of radiosensitivity by histologic examination of biopsy specimens, has necessitated the most careful scrutiny and has encouraged greater accuracy in pathological diagnosis. An increasing knowledge of histogenesis has rendered some of the older terms untenable.

Some confusion still exists because of the diversity of terms used for these various tumors and because of the inherent difficulties in differentiating certain types by either clinical or pathological methods available at present.

However, excellent clinical studies of these tumors are numerous and their varying symptomatologies have been very clearly pointed out. New's worthy article published in 1922 represents the beginning of the renewed interest in the pathology of these regions by the clinicians of this country.

In Paris, Regaud had already observed the peculiar histologic appearance of certain radiosensitive tumors of the pharynx, and in an article published in 1921 by Reverchon and Coutard, Regaud's designation of lympho-epithelioma for certain of these tumors received wider recognition. Apparently, independently, Schmincke in 1921 described a group of tumors somewhat similar to, but not identical with, those named by Regaud. It was thought that the intimate association of endodermal epithelium with lymphocytes was the essential feature in the histologic structure of these tumors. Evidence in favor of this view was the occurrence of tumors presenting this structure in locations in which lympho-epithelium had been previously described by Jolly and by Mollier, namely, the base

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\* By invitation.



of the tongue, the tonsils and the nasopharynx. However, the exact rôle of lymphocytes in these tumors is not at all definitely established, and it is altogether probable that undifferentiation or anaplasia, rather than a special cell of origin accounts for the peculiar structure of these tumors. This seems highly probable since a relatively high percentage of the carcinomas of the base of the tongue, tonsils and nasopharynx are distinctly squamous in character.

The main types of malignant tumors and their relative incidence in various regions about the pharynx can be well illustrated by the statistics furnished in clinical studies from several authoritative sources.

New in 1925 reported a large series of cases with the following distribution and types.

#### CHART I

##### Malignant tumors of the nasopharynx—119 cases.

(1) Epithelioma .....	60 or 50%
(2) Lymphosarcoma .....	42 or 36%
(3) Indeterminate type .....	16 or 14%

##### Malignant tumors of Pharynx—136 cases.

(1) Epithelioma .....	79 or 58%
(2) Lymphosarcoma .....	37 or 27%
(3) Mixed tumors .....	14 or 10%
(4) Malignant, indeterminate type .....	3 or 2%
(5) Sarcoma, special types .....	3 or 2%

##### Malignant tumors of hypopharynx—41 cases.

(1) Epithelioma .....	39 or 95%
(2) Lymphosarcoma .....	2 or 5%

##### Malignant tumors of tonsil—57 cases

(1) Epithelioma .....	41 or 72%
(2) Lymphosarcoma .....	16 or 28%

According to these figures the various types of carcinoma and the lymphosarcomas together constitute 90 percent of all the malignant tumors encountered in the pharynx, nasopharynx, hypopharynx and tonsils, with carcinomas predominating in the proportion of about 2 to 1.

No attempt was made at this time to subdivide the carcinomas, but it had already become apparent that some of the malignant tumors could not be classified with any degree of certainty.

In their outstanding contribution on benign and malignant growths of the nasopharynx, Crowe and Baylor in 1923, placed considerable emphasis on the varieties of sarcomas found to involve the nasopharynx. Some of these arose primarily in the nasopharynx while some encroached upon it from adjacent or nearby regions. The site of origin is often difficult to determine, but in eight of the

twenty-seven cases, the history of early involvement of the eustachian tube suggested that the growth arose in the nasopharynx, while in fourteen cases early nasal obstruction makes a nasal origin probable.

The listed diagnoses for this heterogeneous group of sarcomas are as follows:

#### CHART II

Sarcomas of nasopharynx—27 cases (Crowe and Baylor, 1923).

##### Primary and Secondary

(1) Lymphosarcoma .....	10 or 37%
(2) Angiosarcoma .....	5 or 19%
(3) Myxosarcoma .....	3 or 11%
(4) Round cell sarcoma .....	3 or 11%
(5) Spindle cell sarcoma .....	2 or 7%
(6) Fibrosarcoma .....	1 or 4%
(7) Alveolar sarcoma .....	1 or 4%
(8) Osteosarcoma .....	1 or 4%
(9) No microscopic diagnosis .....	1 or 4%

The varied structures represented by these sarcomas are indicated by the variety of the terms used in their diagnosis. The rates of growth and mechanisms of extension would likewise vary, so that no single group of characteristics would apply to all.

In general, the local growths of sarcomas may become quite bulky, since they grow partly by expansion, and often furnish themselves with a fairly adequate blood supply. The regional lymph nodes are not likely to be invaded by any of the special types of sarcoma, as metastasis is predominantly through the blood vessels, rather than through the lymphatic channels, except in the case of lymphosarcoma.

The relatively benign nasopharyngeal fibromas are likely to be classified as sarcomas if the diagnosis is made from a biopsy specimen alone. These odd tumors which are seen chiefly in males between the ages of ten and twenty-five years are benign only in the sense that they do not metastasize. They grow in part by extension, and therefore, lack a definite capsule; they invade the surrounding tissues and may even destroy bone with which they come in contact. Blood vessels are usually numerous and large in these fibrous tumors, and foci of immature connective tissue cells persist even in hyalinized and scarred areas of the tumor mass. The scattered plasma cells and lymphoid cells in these tumors give a confusing inflammatory aspect to the histologic appearance. According to Bensch, these tumors often cease to grow and gradually tend to disappear after the age of twenty-five years. The growth is thought to originate from

the fibrocartilage of the upper cervical vertebrae, the basilar process of the occipital bone, or the internal lamina of the pterygoid process. The age at which their retrogression is said to occur corresponds with the fusion of the sphenoid with the basilar processes of the occipital bone and the completion of ossification of the cervical vertebrae.

The lymphosarcomas arise from, and reproduce, lymphoid structures. They occur in two common histologic types, which do not vary much in their clinical manifestations, nor in their ready local response to irradiation. The main significance of the two types, is that the reticulum cell type of lymphosarcoma closely resembles some of the diffuse infiltrating carcinomas arising from the epithelium covering the lymphoid tissues of the nasopharynx, tonsils and base of tongue, making an absolute separation difficult or at times impossible.

Quick and Cutler in 1927, introduced the term transitional cell epidermoid carcinoma for a group of radiosensitive intra-oral tumors and reported nineteen cases, eighteen of which occurred in men. The regions of the lingual tonsil, the pharyngeal tonsils and the nasopharynx were considered to be the favorite sites of origin of these transitional cell epidermoid carcinomas.

Just what relationship these tumors held to the lymphoepitheliomas of Regaud and of Schmincke was not made entirely clear. It remained for Ewing in 1929 to attempt the correlation and the differentiation of these groups, and to establish the standards of recognition much as they remain today. A restudy of 100 cases of malignant nasopharyngeal tumors from the records of the Memorial Hospital gave the following revised types and their relative frequencies:

Malignant nasopharyngeal tumors (Ewing, 1929.)

(1) Epidermoid carcinoma—squamous cell type.....	30%
(2) Epidermoid carcinoma—transitional cell type.....	37%
(3) Lymphosarcoma .....	15%
(4) Lymphoepithelioma .....	11%
(5) Adenoid cystic epithelioma .....	4%
(6) Adenoma malignum .....	3%

A comparative study of 200 malignant tumors of the tonsils and the base of the tongue gave the following types and their proportions:

Malignant tumors of tonsils and base of the tongue (200 cases).

(1) Epidermoid carcinoma—squamous cell type.....	72%
(2) Epidermoid carcinoma—transitional cell type.....	12%
(3) Lymphosarcoma .....	9%
(4) Lymphoepithelioma .....	4%
(5) Unclassified .....	3%

According to the above findings, the sarcomas recognized constituted only 15 percent of the malignant tumors of the nasopharynx, while the various types of carcinomas totalled 85 percent. Apparently most of the unusual tumors were epithelial in type and were classed as adenoid cystic epitheliomas or adenoma-malignum.

Probably some of these were of mixed tumor type, resembling the glandular areas of the more frequent salivary gland tumors, also not infrequently found in the palate, lachrymal glands and occasionally elsewhere. All of the sarcomas listed are classed as lymphosarcomas, including both of the commonly recognized types, the lymphocytic and the reticulum cell.

The involvement of the cervical lymph nodes is the most frequent localization of lymphosarcoma. Some of these cases begin with a swelling of the lymphoid tissue and diffuse infiltration of the mucosa of the pharyngeal wall or of the tonsillar ring. Ulceration soon occurs and remains a persistent or recurrent complication. The process extends to the cervical lymph nodes, the nares, base of skull, orbit, or cranial cavity. In the advanced stages, the tissues of pharynx, face and neck may become fused in a bulky infiltrating tumor which compresses the vessels without rupturing into them, and causes varied symptoms from involvement of vessels, nerves, trachea and special sense organs.

Many of these cases terminate without any marked extension to other regions, while others spread progressively to other groups of lymph nodes and other lymphoid tissues. The frequent involvement of the lymphoid tissues of pharynx, tonsils and cervical lymph nodes suggests that some irritating agent entering these tissues precipitates the malignant proliferation of the labile cells of these lymphoid tissues.

It should be noted that endotheliomas are not recognized among the primary tumors of the nasopharynx, tonsils or base of tongue, or at least, that they did not occur among the 300 tumors reported in Ewing's critical study. Their differentiation at present from some of the transitional cell carcinomas would be impossible by available histologic methods. Furthermore, a diagnosis of endothelioma of the cervical lymph nodes could be substantiated only in case no primary tumor is found in mouth, nose, or pharynx to account for an endothelial-like tumor in the cervical lymph nodes. This prerequisite has greatly reduced the frequency of the diagnosis of endothelioma in any of these regions and makes very doubtful all such diagnoses made rather frequently in this region previous to the past few years.



As has just been pointed out in the case of the nasopharynx, epithelial tumors, likewise, constitute the great bulk of malignancies, involving the tonsils and base of the tongue, making up a total of 88 percent in the statistics cited.

Only 9 percent of this group were classed as sarcomas and all of these were diagnosed lymphosarcoma although an additional 3 percent of these tumors were not definitely classified.

It is of interest to note that squamous cell carcinomas occurred less frequently than transitional cell carcinomas in the nasopharynx while a ratio of six of the former to one of the latter was found in tumors of the tonsils and the base of the tongue.

#### THE TYPES OF CARCINOMA OF THE NASOPHARYNX

As elsewhere in the body, these carcinomas reproduce more or less perfectly the epithelium from which they take their origin, and the grade of potential malignancy roughly parallels the lack of differentiation of the tumor cells. In the more anaplastic tumors it becomes increasingly difficult to determine the cells of origin and even their epithelial origin may be obscured.

It is assumed that most of the carcinomas of the nasopharynx and tonsillar region arise from the stratified squamous or pseudostratified columnar epithelium covering these mucous surfaces.

There is, however, a widespread belief that the epithelium covering the underlying lymphoid tissues of nasopharynx, tonsils, tongue and elsewhere possesses some inherent qualities which modify its behavior in the tumors of these regions. This conception has been responsible for much of the more recent restudy of these tumors.

(1) *Epidermoid Carcinoma-Squamous cell type*.—These carcinomas retain some of the squamous cell characteristics in the tumor cells, as indicated by intracellular cornification and hyalinization, the formation of intercellular bridges, and occasionally epithelial pearl formation.

The carcinomas of this group are quite similar to the squamous cell carcinomas of the skin, tongue, esophagus and various other mucous surfaces covered by stratified squamous epithelium. They occur in the nasopharynx in varying degrees of anaplasia, but most of those definitely recognized as such are fairly well-differentiated and are usually classed as Grade I or Grade II malignancies.

The primary focus of squamous cell carcinoma in the nasopharynx is said to present a coarsely granular surface which usually progresses to ulceration, giving an elevated border with a depressed ulcer crater.

However, the gross lesion cannot be differentiated with any degree of certainty from those of other carcinoma types.

In the more differentiated squamous cell carcinomas, metastases occur late in the cervical lymph nodes.

(2) *Transitional Cell Epidermoid Carcinoma*.—Originally the transitional cell carcinoma of the nasopharynx, tonsils and base of tongue was formed into a separate group because the tumor cells lacked the usual features of squamous cell carcinomas and because the tumor cells displayed an unusual susceptibility to irradiation. The term transitional cell indicates a structure intermediate in appearance between squamous cell, on the one hand, the basal cell, on the other, although the similarity to squamous cell type is actually much closer, and it seems likely that the transitional cells are often only anaplastic squamous cells.

The name applied to this group of tumors has a structural significance, and it is not dependent upon any peculiar specialization of the epithelial cells from which the tumors develop. In other words, it has a more general application than the term lymphoepithelioma as used by Regaud.

Histologically, the tumor cells are relatively small, with large hyperchromatic nuclei, and scanty cytoplasm, and many mitotic figures. The cells are closely packed with delicate connective tissue stroma. The tumor cells may form solid masses or they may grow more diffusely in anastomosing columns or cords. Flat, pavement characters, intercellular bridges, cornification and pearl formation are regularly absent.

The primary focus when found is said to present a finely granular, velvety surface which looks more like an erosion of the mucous surface than a true ulceration. It appears flattened and gives the impression of having originated in the deeper structures and later adhered to the mucous membrane. Cutler states that in some early cases, there has been neither erosion nor ulceration, but rather the appearance of fixation of the mucous membrane over a mass beneath it.

Early metastasis to the cervical lymph nodes is one of the most constant and characteristic features of transitional cell carcinoma of these regions. In only a few early cases has it been absent when the patient was first observed. The location of the metastases varies somewhat, but they usually appear under or in front of the sternomastoid muscle and extend upward toward the angle of the jaw. At first unilateral, except when the primary focus lies directly in the midline, the adenopathy soon extends and involves the opposite site of the neck. The lymph nodes remain rather freely movable

until the late stages of the disease, when they may become fused into a bulky mass, exerting marked pressure symptoms. Visceral metastases are not infrequent with transitional cell carcinomas; metastases have been described in liver and in bones.

Tumors of this type occur occasionally in young persons under twenty years of age, although they occur also in the later age periods. At first most reported cases were in males, but subsequent reports include a larger proportion of females.

Most of the transitional cell carcinomas, on a histologic basis, are classed as having a high potential malignancy and listed as Grades III and IV.

(3) *Lymphoepithelioma*.—The term lymphoepithelioma as used by Regaud and by Schmincke is based upon the assumption that a definite interrelationship exists between the epithelium covering the lymphoid tissues of the pharynx and the underlying lymphocytes. No definite specialization of this epithelium is generally accepted as proven, although such a possibility cannot be entirely set aside. A definite line of division between transitional cell carcinomas and lymphoepitheliomas is difficult to establish, and no uniformity prevails at present, throughout the world, in the separation of these two types. Ewing states that Doctor Laccassague of the Pasteur Institution (1929) in Paris included under the term lymphoepithelioma, the various tumors here designated as transitional cell carcinomas.

However, a small group of carcinomas of the nasopharynx and of the tonsils present structures which coincide with the tumors described by Regaud and by Schmincke as lymphoepitheliomas. The epithelial cells form wide sheets or narrower anastomosing cords which extend into or diffusely invade the surrounding lymphoid tissues.

The cells are large and lightly stained, with large vesicular nuclei and prominent nucleoli.

In places, the lymphocytic infiltration is so marked as largely to obscure the tumor cells.

The structure strongly suggests primary endothelioma and is often difficult to distinguish from the reticulum cell type of lymphosarcoma.

The persistence of many lymphocytes in both the primary and secondary growths, together with the large vesicular tumor cells, are taken as the essential histologic characteristics for the diagnosis of lymphoepithelioma.

These tumors occur at all ages, but are especially frequent between thirty and sixty years.

They produce rather small local growth which are soft, slow to ulcerate and frequently overlooked. Like the transitional cell carcinomas they produce early metastases to the cervical lymph nodes, unilateral or bilateral, and ultimately may give systemic metastases.

It seems doubtful if any definite clinical advantage is secured by the separation of this group of tumors from the larger group of transitional cell carcinomas.

### SUMMARY

(1) It is apparent that ready response to irradiation on the part of several groups of these tumors which occur in the nasopharynx and elsewhere, has been largely responsible for the renewed interest in the histologic structure and the reclassification of these neoplasms. In this region, the radio-sensitivity of the groups of carcinomas roughly parallels the degree of anaplasia of the tumor cells. However, other factors alter the response to irradiation and histologic grading of potential malignancy must not be confused with "radio-sensitivity grading."

(2) Occasional rare types of sarcoma are encountered, but most of the tumors of non-epithelial type are classed as lymphosarcomas, and closely resemble similar tumors found elsewhere in the body.

(3) The carcinomas are varied in structure and must be subdivided into several important groups. The epidermoid carcinomas form fairly well-defined squamous cells and transitional cell types.

(4) The term lymphoepithelioma is reserved for tumors whose epithelial properties are not definite and whose tumor cells are constantly associated with abundant lymphocytes, in the stroma of both primary and secondary growths.

(5) An occasional glandular carcinoma may arise from the mucous glands of the lining mucous membrane, and a few comparatively benign mixed tumors occur in the nasopharynx.

(6) Remnants of the hypophyseal duct of Rathke's pouch account for occasional tumors of the adamantinoma type; while remnants of the chorda dorsalis or notochord, may be responsible for a few of the peculiar tumors invading the nasopharynx.



## CANCER OF THE LARYNX, TRACHEA AND BRONCHIAL TUBES

By M. F. ARBUCKLE, M.D.

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With increasing knowledge of the nature of laryngeal, tracheal and bronchial cancer and with improved methods of treatment, the outlook for such patients is now much brighter. The dissemination of information among the laity as well as the medical profession regarding the early symptoms of cancer of the upper respiratory tract is accomplishing much towards early diagnosis, which is, of course, so very important a consideration.

In view of the unfortunate lack of symptoms of a kind which drive patients to see a doctor in the early stages of this disease, such as pain, constant effort along educational lines is extremely important. All experienced observers have seen instances in which patients suffering with cancer in this region have presented themselves for initial examination only when the disease was already far advanced. The almost total lack of pain, bleeding or other evidence of disease in cancer of the lower respiratory tract in the early stage is a noteworthy fact.

In endobronchial cancer the early symptoms are of a kind which may be ascribed to any one of a number of disturbances in this region. Probably the earliest and most constant symptom is cough. This, like all other symptoms of cancer of the lung, is rather characteristic when carefully studied. It is a persistent, irritating, wracking cough not relieved by any remedy or treatment. At first it is non-productive and painless, but later mucopurulent sputum streaked with blood is brought up in gradually increasing amounts and pain occurs with greater severity. As the mass grows with consequent encroachment on the bronchial lumen, the results of obstruction become apparent. At first they are those of interference with lung drainage and ventilation, *viz.*, "drowned lung," then atelectasis and infection with abscess formation; later, pleural effusion which may be bloody and often contains cancer cells. Still later, empyema may occur, and with it invasion of the thoracic walls by the cancer through direct extension. A very useful method of studying bloody fluid from the pleural cavity consists of centrifuging the fluid and imbedding the sediment in paraffin and sectioning it like any pathological specimen.

Occasionally the first symptom of cancer of the lung may be an unexplained hemoptysis, or an atelectasis of a portion of the lung or other symptoms of intracranial disease such as convulsions which, when carefully studied, are found to be due to metastasis to some other part of the body, constituting the first symptom to call attention to the lung lesion.

Improved methods of diagnosis such as direct examination and biopsy, together with the application of roentgen study aided by the use of radiopaque oils, enable us to arrive at a definite conclusion in a large percentage of cases.

Histopathologic study of tissue specimens with special regard to the cellular constitution or histogenesis of the tumor and its probable degree of radiosensitivity has led to great advances in the methods employed in the treatment of this disease. For this reason bronchoscopic examination should be carried out in every case of unexplained bronchial obstruction with symptoms such as above enumerated.

With increasing experience it is evident that endobronchial cancer is much more frequent than it was formerly thought to be, also that it may occur much earlier in life.

Thomson and Colledge's book, "Cancer of the Larynx" affords us a most helpful guide in the classification and subsequent recommendations for treatment.

Complete surgical removal, when possible; seems to be the method of choice in dealing with cancer, and if Sir St. Clair Thomson's recommendations are carefully followed, it will prove to be a most satisfactory procedure.

As a result of the great amount of work being done all over the world in the field of radiation, it is possible that our ideas regarding this remedial measure may take on an entirely different aspect within the comparatively near future.

In cancer of the trachea complete surgical removal is, of course, out of the question, and we must depend entirely upon some other remedy. Radiation probably is the one of choice and while this type of case is rare, the necessity for intervention is obvious when it does occur.

In endobronchial cancer, the plan of attack must necessarily depend upon many factors. The location of the tumor will determine whether or not we may be able to obtain a biopsy specimen. In our experience about 40 to 45 percent have been beyond the range of visibility. As has been shown by Womack and Tuttle, there is evidently a very great difference in the rate of growth and

spread of the parenchymal type of tumor as compared with that of the hilar tumor.

As is generally known, surgical interference for cancer of the lung has received great impetus during the past few years. Naturally this method may be employed with hope of success only in cases in which the tumor is favorably located and still well localized. The proximity of the lesion to the bifurcation in cancer of the larger tubes can be made out with a fair degree of accuracy by the bronchoscopist. This information combined with that obtained by careful physical examination and roentgen study should enable us to discover metastasis or direct extension to neighboring parts if present.

Radiation for this type of tumor thus far has proven disappointing, but with further developments and improvements in technic it may be possible to work out a method of radiation applicable in such cases before metastasis to distant parts has occurred.

# THE UTILIZATION OF SUSPENSION APPARATUS IN MALIGNANCIES OF THE LARYNX AND DEEPER STRUCTURES OF THE LOWER PHARYNX AND ESOPHAGUS

By F. E. LEJEUNE

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The utilization of suspension laryngoscopy in the approach to malignancies of the larynx and deeper structures of the lower pharynx and esophagus is a well-known procedure. In early malignancies of the larynx, suspension laryngoscopy should certainly be the method of choice. In lesions of the hypopharynx it is a distinct aid not only in determining the exact extent of the lesion, but in the surgical attack or the application of electrocautery or radium. Malignancies of the esophagus limited to its upper borders can be well observed by means of suspension laryngoscopy.

In order that intralaryngeal surgery be successfully accomplished, an early diagnosis in malignant lesions is imperative. The removal of benign growths by intralaryngeal surgery under suspension laryngoscopy is accomplished with accuracy, thoroughness and ease. Malignant conditions in their early stages may be successfully attacked by endolaryngeal surgery. The skill of the laryngologist will be an important factor in the use of the suspension laryngoscope. While excellent exposure will facilitate the operative procedure, the final results of endolaryngeal surgery are largely dependent upon the thoroughness of the surgical attack and the extent of the tumor mass. In the intrinsic group of carcinomas in which the lesion is limited to definite areas, three types of lesions are recognized when considering endolaryngeal surgery. Cordal lesions, limited to the anterior and middle third of the vocal cords, in which the growth is practically a surface lesion and consequently limited to the cells above the basilar membrane, represent the earliest type of malignancies. The symptoms produced by such a small tumor mass as this type represents are indeed slight and only mild voice changes may be noted. Such a lesion as described may exist for a long period of time as is evident by observation and record. The operative procedure under suspension involves the resection of the tumor mass with a free margin of uninvolved tissue on all sides. In the earliest type of cases, it is desirable, if possible to remove the affected cord from near the anterior commissure to a point just anterior to the



vocal process. Preservation of the vocal process, possible only in early malignancies, is responsible for good movement and voice, when in due course of time a pseudo-cord is manufactured. I do not use the punch forceps in removing any laryngeal growth. My observation and experience coincide with that of Foster, who states that more rapid and better healing results when the tumor is grasped and pulled to the median line and removed by a straight incision. This applies principally to benign growths.

The second group is an extension of the first with an increase in cell proliferation, induration, size of tumor and lagging of the vocal cord, but without fixation. The lagging indicates that cell proliferation and growth have extended sufficiently to involve the muscle fibers, but with no involvement of the joint. Complete extirpation of the cord with a wide free margin, followed by surgical coagulation of the base is advocated and practiced in these cases. New has determined from careful studies that extension of malignant cells was found at variable distances beyond the visible growth dependent upon the type and grading of the malignancy present. Coagulation is used after surgical removal of the growth for the purpose of destroying any malignant cells which might have extended beyond the line of incision. Preoperative irradiation is used routinely in all cases falling into this particular class. Following operative procedure irradiation is again instituted. This combination of surgery and irradiation has given me the best results so far. In all such cases the patient should remain under observation, as any evidence of a recurrence requires immediate attention. I have a patient who required three intralaryngeal operations in order to control a squamous cell carcinoma growing on one vocal cord, the last operation having been done three and one-half years ago. Today the patient is apparently well and only a hoarse voice produced by a pseudo-cord remains as evidence of former trouble.

In group three we find those border line cases where the progress of the lesion is such as to make the success of endolaryngeal surgery doubtful. These types of cases are best suited for laryngofissure and should be taken care of in that manner.

Early growths occurring on the vocal cords can be removed by endolaryngeal surgery with the same degree of success offered by laryngofissure plus the additional advantage of preserving the continuity of the larynx. The preservation of the integrity of the larynx, in my opinion, is very important and can only be accomplished when we are fortunate enough to see these malignant growths in their early stages. Suspension laryngoscopy offers an excellent approach to

tumors of the hypopharynx, pyriform fossa and mouth of the esophagus. As a rule, however, little can be accomplished in these cases by endolaryngeal surgery. Malignant tumors of the lateral wall of the pharynx, epiglottis and aryepiglottic fold develop more slowly and metastasize later than those tumors occurring in the pyriform fossa and lowest part of the hypopharynx (Von Eicken) which develop more rapidly and with earlier metastasis. Unfortunately malignant growths of the hypopharynx and pyriform fossa give little warning of their presence until the lesion is well extended.

The complaint of a sensation of fulness or consciousness of the lower pharynx should never be dismissed without a careful examination. Tumors of the lateral wall of the larynx and pyriform fossa are easily seen with the laryngeal mirror. Carcinomas of the post-cricoid region and the lower pharynx present more difficulty in diagnosis. The prognosis in surgical intervention for carcinoma of the hypopharynx and pyriform fossa is unfavorable. Occasionally in well selected cases with limited involvement some success is attained. A squamous cell carcinoma, grade 2, involving the upper portion of the epiglottis was removed by low amputation followed by extensive electrocoagulation and remains well without recurrence four years after operation. A supraglottic tumor with a pseudocapsule attached to the right aryepiglottic fold and a portion of the pyriform sinus was dissected out completely and base coagulated. Microscopic examination showed grade 3, squamous cell carcinoma. At no time were there any palpable glands. Fractional radiation was instituted and patient remains well three years after operation.

In those puzzling cases where one or several smears are desired for examination or where biopsy is necessary, suspension laryngoscopy is unexcelled. I find great comfort when dealing with a questionable laryngeal lesion in requesting the presence of the pathologist, who may also sit at the head of the table and deliberately inspect, palpate and render an opinion as to the lesion in question.

My experience with malignancies of the esophagus has not been attended with much success. With the development of radiation a ray of hope is offered these unfortunates as well as those whose lesions are confined to the hypopharynx and pyriform sinuses.

# IRRADIATION THERAPY OF MALIGNANT TUMORS OF THE ORAL CAVITY, EYE, EAR, NOSE AND THROAT

By CHARLES L. MARTIN, M. D.

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Cancer therapists have for years dreamed of a remedy possessed of the power to relentlessly track down and destroy every cancer cell in the body without damaging the normal tissues. When radiant energy was first shown to exert a more harmful effect on certain neoplastic cells than on the adjoining normal structures our hopes were aroused and the desired medium seemed to be at hand. However, clinical experience soon revealed a lack of regression in many of the malignant tumors treated with the earlier technics. These new powerful rays did not seem to have a selectivity great enough to save the normal structures when doses capable of killing the less malignant types of cancer cells were given. It became evident that unless this selectivity could be improved the uses of the new method would be much restricted. Progress has been slow and technics will no doubt be definitely improved in the future, but the field of irradiation therapy has been broadened by the evolution of the following principles:

(1) A total of three to twelve minimum erythema doses of irradiation is needed for the complete extermination of the various grades of epidermoid carcinoma.

(2) Recovery is more rapid and the surrounding tissues show a quicker return to normal when the treatment is administered over a prolonged period (usually seven to twenty-one days, at present).

(3) An attempt should always be made to produce a cure with the first series of treatments, since the second attempt is never so effective.

(4) Short wave lengths produced with heavy filtration lessen the injury to surrounding normal tissues, but this protection is not essential in the treatment of superficial lesions.

(5) Infections and other sources of irritation definitely retard healing after irradiation.

## TECHNICS

The earliest therapy was done with x-rays having a relatively long wave length and low penetrating power, and naturally the good results were limited to malignant tumors of the skin. Pusey,<sup>1</sup>

who had no accurate measuring devices available, published case reports with illustrations showing patients cured of squamous cell cancer of the lip more than twenty-five years ago. Although his technic utilized many of the principles elaborated above, it has never been popular with dermatologists. However, it is still a very valuable procedure, and for superficial tumors measuring an inch or less in diameter it may still be considered the method of choice. It will hereafter be designated as the "superficial x-ray technic." The factors used by us are 87 kv. peaks, 5 ma., a target skin distance of 10 inches, and a filter of 0.5 mm. of aluminum which with our apparatus produce a mild erythema over an area one inch square with an exposure time of five minutes. The size of the area treated is of course quite important, since this dose produces 555 r with back-scattering over a circular area  $\frac{3}{4}$  inch in diameter, 640 r over an area  $1\frac{1}{2}$  inches in diameter, and 666 r over an area 2 inches in diameter. In practice one or two erythema doses is given at each visit and the treatments are repeated every second day until a series of six or more is administered.

The shorter wave lengths for deep therapy were first generated at 130 to 140 kv. peak with filters of 3 or 4 mm. of aluminum. Just after the World War, German physicists developed machines generating 200 kv. peak and advocated filters of  $\frac{1}{2}$  to 1 mm. of copper. Elaborate charts were produced showing exactly how much radiant energy could be delivered to various points within the body and complicated plans for cross-firing were worked out. All of these changes tended to improve the therapy of deep seated tumors, but unfortunately the theory was advanced that the delivery of a single erythema dose to all parts of a malignant tumor by the new method would cause its complete regression. Although no clinical evidence was ever produced to support this theory, the single "massive dose" plan has been carried out in many American clinics ever since its introduction from abroad.

In 1925, Pfahler proposed his saturation method, which constituted an important step forward in the evolution of efficient depth dosage. He adopted the theory of Kingery, which states that a dose of x-rays decays in accordance with the values indicated by a logarithmic curve. An erythema dose was rapidly built up in a few days and small doses were then administered every few days thereafter. These smaller doses were calculated from the curves and were supposed to be just large enough to return the decaying dose to its original level. This plan really succeeded in delivering about  $2\frac{1}{2}$



erythema doses to a single area in one month and was therefore a definite improvement on the massive dose method.

There has been a tendency in this country to build larger machines with higher voltages in an effort to obtain shorter wave lengths and thereby improve the selective action of the rays. Such a machine operating at 700 kv. peak has been in use at the Memorial Hospital in New York since 1931, and a few others have been installed elsewhere, but although the results obtained indicate some increased efficiency they have not as yet seemed to justify the huge expense involved.

The most important advance in deep therapy has come from France, where it has been shown, both experimentally and clinically, that very large doses of x-rays may be passed through normal tissues without irreparable damage, provided they are properly fractionated and prolonged over a sufficient period of time. Experiments performed by Regaud and Ferraux<sup>2</sup> with rams' testicles show that although the fractionization spares the adult cells of the normal tissue, the effect on the more embryonic cells is almost the same as it would have been if the dose had all been given at once. The increased selectivity produced by this method has been utilized clinically by Coutard<sup>3</sup> for a period of about fourteen years, and his results testify to its efficiency. Although Coutard believes that a low intensity (6 r per minute) is essential to the success of his method, good results have been obtained in this country with much higher intensities by Martin and McNattin<sup>4</sup> and the author.<sup>5</sup>

Radium technic has advanced in a somewhat similar fashion. Interstitial work was first done with relatively large amounts of the salt in needles having thin walls of steel which allowed many of the more irritating rays to pass through. Regaud<sup>6</sup> began to advocate heavy filtration, low intensity and long periods of radiation some fifteen years ago. In 1926, Failla<sup>7</sup> described the gold radon seed which carries out Regaud's principles except that the filtration is not maximum. These implants are advantageous because of their small size and because they may be left permanently in place. Unfortunately their preparation requires a very expensive set of equipment. In 1927 Evans and Cade<sup>8</sup> advocated radium element needles that are both economical and efficient. They may be used as a substitute for radon seeds which radiologists in Texas obtain only from New York City. These needles are made of platinum-irridium and their dimensions are given in Fig. 1. Details relative to their use have been published in previous articles.<sup>9, 10</sup> It has been shown that a cubic centimeter of squamous cell carcinoma will regress if 0.6 mg. of radium

filtered with 0.5 mm. of platinum be placed at its center for a period of seven to eight days, and for this reason all of the needles contain 0.6 mg. of radium per centimeter of active length.

It is obviously impossible to discuss the detailed use of the above technics in the treatment of all the many types of malignant tumors occurring about the head and neck in a paper of this sort, and a few of the regions yielding the most promising results will, therefore, be singled out.

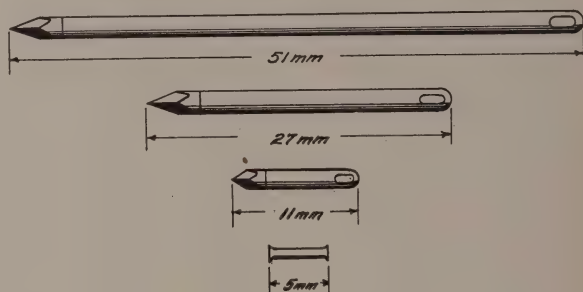


FIG. 1. Relative sizes of weak platinum needles and a radon seed. The needles contain 0.6 mg. of radium per cm. of active length and have wall thicknesses of 0.5 to 0.6 mm. The seeds are made of gold or platinum and have walls 0.3 mm. thick.

### CANCER OF THE LIP

Cancer of the lip has in our experience proven a very favorable lesion for radiation therapy. When the tumor measures no more than one inch in diameter it is treated by the superficial x-ray technic because of the superior cosmetic results that may be obtained. The treatments are administered to the growth and a narrow margin of adjoining tissue on alternate days for a period of about two weeks, and a total of six to twelve erythema doses is given, the variation in dosage depending upon the thickness of the tumor. A severe reaction follows this procedure, but healing is usually complete in six to eight weeks, and the lack of scarring is evident in the cases illustrated in Fig. 2. When the tumors are large and extend into the cheek at the corner of the mouth, or when they are deeply indurated, the weak radium needle technic described under the heading of intra-oral cancer is used. The care of the neck containing adenopathy will be discussed later on, but our experience has led us to the conclusion that a routine block dissection is not necessary when no glands are palpable. In 1931, J. M. Martin<sup>11</sup> reported a series of 108 cases treated by the above method between 1906 and 1925. None of these patients had palpable glands at the time of treatment, and 104 of them had remained well for periods of five years or more. Surgical



FIG. 2. Squamous cell carcinomas of the lip treated with the superficial x-ray technic which have remained well for periods varying from four to six years without neck surgery. Note the absence of scarring after healing occurs.

results covering routine block dissections are certainly no better, and it seems difficult to avoid the conclusion that most operations in cases of this sort are unnecessary.





FIG. 3. Large squamous cell carcinoma Grade II which grew in the left cheek and pushed the molar teeth out of their sockets. The radium needles shown in the radiograph remained in place seven days and were augmented by 1,500 mg. hours delivered externally with 50 mg. radium packs and a single erythema dose of deep x-rays delivered to the side of the neck. There is no palpable or visible evidence of carcinoma made out at the end of two years and three months, at which time the lower photograph was made.

### INTRAORAL CANCER

Tumors involving the cheeks, alveolar margins, anterior tongue and floor of the mouth are suitable for interstitial radium therapy because of their accessibility. Radon seeds are quite efficient in these regions, but the heavily filtered weak radium element needles are also very useful. It is desirable to place these needles beneath the tumor bed and beyond the growing edges, spacing them about 1 cm.



apart. Each needle is sutured in place and a guy thread is brought outside the mouth and attached to the cheek so that it cannot be lost or swallowed. Most implantations are done under local anesthesia. After the work is completed radiographs are made in order that the needle pattern may be studied. When the radium distribution is not correct the needles may be easily rearranged. In most cases the treatment is continued for seven days, during which time it is necessary to pay strict attention to oral hygiene. The mouth is irrigated frequently with warm bland solutions and the involved area is painted daily with 5 percent mercurochrome. Use of a spray containing liquid albolene at regular intervals is desirable. Pain is usually not a troublesome symptom after the first twenty-four hours. Following removal of the needles the tumor usually melts down rapidly and a white membranous reaction appears. This change is a superficial one and is in no way similar to the painful sloughs formerly produced by steel needles. The membrane clears away after a few weeks, leaving a clean granulating surface, and healing occurs in about two months. A soft pliable scar which interferes very little with function results. In tongue lesions taste is not interfered with and mobility is unimpaired. Bony structures situated near the sources of radiant energy show little or no damage, although occasionally flakes of the cortex may separate. Large tumors involving the hard palate, such as the one shown in Fig. 4, have disappeared promptly without any apparent bone damage. A review of our rather limited material was published in a recent article.<sup>12</sup> All of the primary lesions, except a few in which local irritation played a big rôle, showed satisfactory regression. Forty percent have remained well for one or more years. Since our experience with weak radium needles is of only about four years' duration we can provide no five-year statistics as yet.

#### CANCER OF THE EYE

Malignant tumors frequently appear on the eyelid and at the inner canthus, where it is very important to preserve functions. Fortunately most of such lesions are very radiosensitive, and the main problem is the protection of the normal structures of the eye. Both the conjunctiva and the lens can be seriously damaged by large therapeutic doses. The superficial x-ray technic produces rays of minimum penetrating power and it is, therefore, best suited for this work. At each visit an oval lead shield is slipped under the cocaineized lids, after which the lesion is isolated with other lead shields and inten-

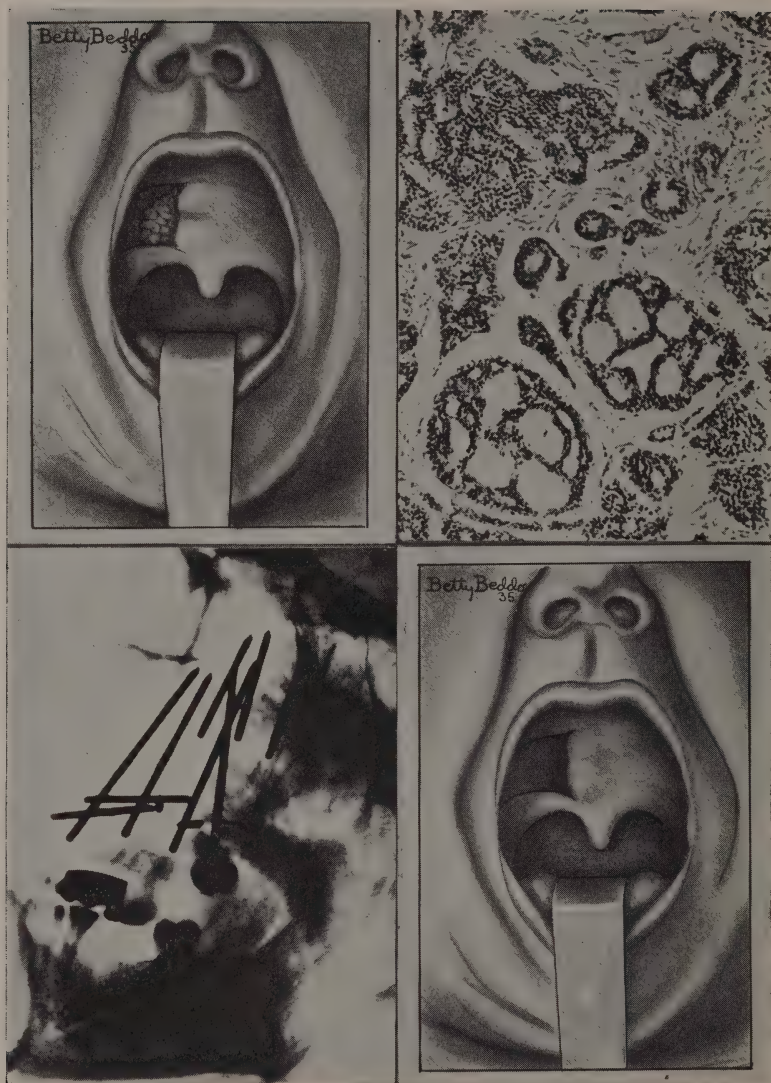


FIG. 4. Recurrent adenocarcinoma Grade I of hard palate which recurred both in the palate and in the antrum following operation as shown in the upper drawing. The lower sketch shows the absence of all visible evidence of the tumor four months after the radium needle implantation shown in the radiograph. The needles remained in place for seven days.

sively radiated. When healing occurs all of the normal tissue is conserved and usually no cosmetic surgery is necessary. Tumors near the nose frequently extend inside the orbit, where masses of considerable size can be palpated just inside the orbital rim. These extensions can be satisfactorily treated by the insertion of the 1.33 mg.

radium needles at 1 cm. intervals for a period of seven days. No eye damage results if the needles are planted near the wall of the orbit.

The superficial x-ray technic is particularly well suited to the treatment of malignant tumors of the ocular conjunctiva. Grier<sup>13</sup> reported eleven such cases successfully treated with x-rays ten years ago. At each visit it is our custom to fit a carefully prepared lead shield over the cocainized eyeball with lid retractors in place. A hole is cut in the shield to fit the lesion and an erythema dose is administered every second day until six such doses are given. The reaction is slight and no eye damage results. Photographs of a large carcinoma of this type before and after treatment were published in a recent article.<sup>14</sup> This patient has been well for more than two years and his vision is normal on the treated side.

It is our feeling that radiation has no place in the treatment of primary intra-ocular tumors. However, the Coutard plan of administering deep therapy is of definite value as a postoperative procedure, particularly when metastases seem likely.

#### CANCER OF THE EAR

Small superficial carcinomas of the skin of the ear respond well to minimum total amounts of superficial x-ray therapy. However, the use of large doses in this region is hazardous because a very stubborn and painful reaction is frequently set up. This peculiarity has been attributed to the effects of radiant energy on cartilage. It seems best, therefore, to treat well advanced lesions by complete removal of all or part of the ear. Contrary to the belief of many surgeons, this procedure is frequently not curative, since deep invasion often extends down into the tissues about the parotid and below the auricle. It is our custom to remove the auricle, after which weak radium needles are placed around the ear and in all suspicious places for a period of a week. A good result obtained in this way is shown in Fig. 5. When massive tumors involve the ear and the side of the face, doses ranging between 4,000 and 5,000 r administered during a period of approximately three weeks, by the Coutard plan, often produce striking improvement. The number of postoperative cases of cancer of the ear with deep metastases that have appeared in our clinic make it evident that more attention should be given this subject.

#### CANCER OF THE PHARYNX AND LARYNX

With the advent of interstitial radium therapy various plans were worked out for implanting the relatively inaccessible tumors



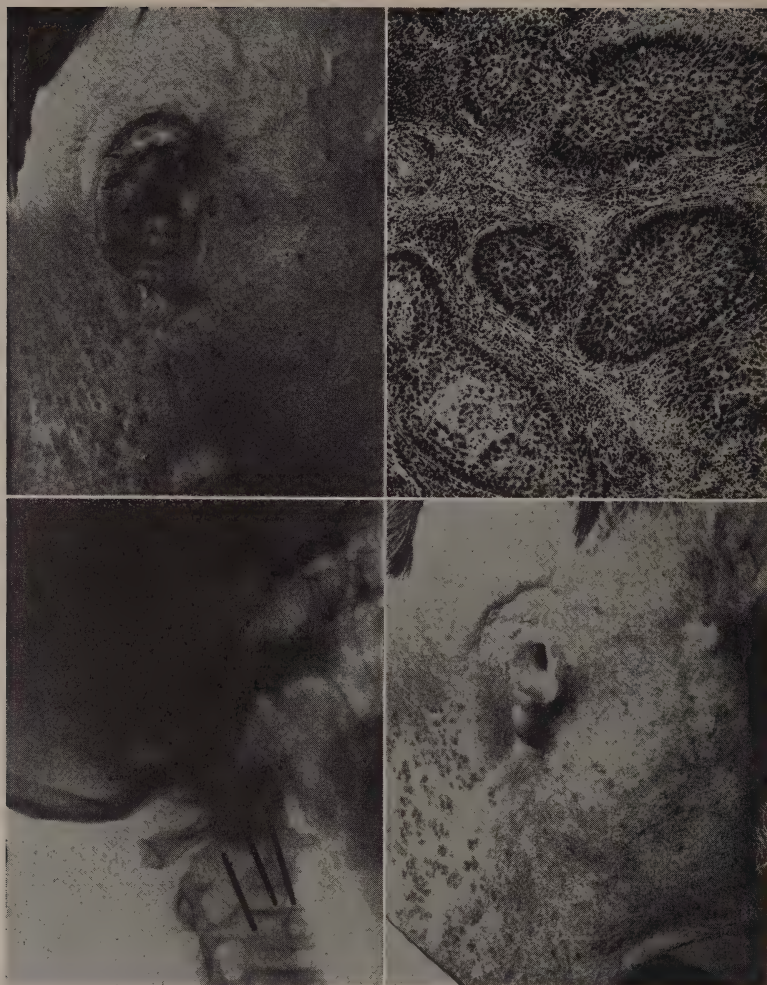


FIG. 5. Squamous cell carcinoma Grade III of the upper auricle with deep involvement of the surrounding tissues treated with radio knife resection and the implantation of weak radium needles for seven days. There was a palpable gland in the neck. The lower photograph made one year and two months later shows perfect healing. No palpable evidence of malignancy could be made out.

of the pharynx and larynx, and radon seeds were obviously better than needles for this type of work. Some excellent results were obtained, but much difficulty was experienced in evenly irradiating the larger tumors and untoward reactions in the cartilage of the larynx limited the dosage in this region. The internal irradiation was augmented by radium packs and a massive dose x-ray technic, but the external irradiation did not assume a rôle of major importance until Coutard elaborated his method whereby large, efficient



doses could be delivered to the midline of the throat without the production of serious injuries to the normal tissues. This made it possible to rely on deep x-rays alone, and the present tendency is to eliminate implanted radium in the treatment of the deeply seated tumors.

Fortunately the carcinomas of the pharynx are much more radiosensitive than their histology would indicate, and they do not require doses as large as those needed for the epithelial neoplasms of the mouth. Coutard felt that he obtained his best results with 190 kv. peak, 2 mm. of zinc filter, 5 ma. of current and a target skin distance of 50 cm. Areas measuring not more than 15 cm. on a side were laid out on the lateral aspects of the neck overlying the involved region and treated on alternate days. Each dose ranged from 200 to 300 r measured in the beam without back scattering, and the treatment was continued for three or four weeks. The total dosage over each skin area amounted to 3,000 r to 5,000 r, and about 3,500 r was delivered to the tumor. As the treatment progressed the throat became very sore and finally a superficial white membrane appeared on the surface. As the mucous membrane reaction subsided the skin surfaces became red and finally much of the epithelium disappeared in the treated areas leaving a moist surface on which small patches of epithelium often remained. This reaction also healed readily, so that there were few sequelæ present after two months had elapsed. The malignant tumors melted away as the reaction in the throat appeared, and many of them were permanently controlled. Between 1920 and 1926 Coutard treated 212 malignant tumors of the pharynx and larynx, many of which were inoperable, and 20 percent of this group remained symptom-free for a period of five years.

About five years ago Lenz, Coakley and Stout<sup>15</sup> began the treatment of a series of thirty-one cases of cancer of the pharynx and larynx, using 200 kv. peak, 8 ma., 1.86 mm. of copper and 1 mm. of aluminum filter and a target skin distance of 50 cm. Fields were laid out, usually on the sides of the neck, and daily doses of 400 r to a single area were given until each of them received from 2,800 to 4,000 r. Approximately 45 percent of these cases remained clinically well for periods varying from nine months to twenty-four months of observation.

In 1931 Martin and McNattin adopted a technic utilizing 200 kv. peak, a filter of 0.5 cm. copper and 2.5 al., 4 to 30 ma. of current and a target skin distance of 50 to 60 cm. Malignant tumors of the tonsil, soft palate, pharyngeal walls, nasopharynx, extrinsic and intrinsic larynx, base of the tongue and floor of the mouth were

treated. Circular parts, 7 cm. in diameter, were used for most of these patients. Out of a series of 140 cases, 29 percent have remained free of disease for periods varying from  $1\frac{3}{4}$  to  $2\frac{1}{2}$  years. In a few cases the treatment was augmented by radon seeds implanted in metastatic cervical glands.

For  $1\frac{1}{2}$  years we have used a technic similar to that of Martin and McNattin. Our enthusiasm was aroused when we observed the rapid disappearance of a very large prickly cell carcinoma of the face after it had received 3,600 r in twelve equal doses given over a period of fourteen days. An effort has been made to deliver somewhat similar doses to tumors in the throat, and the immediate results have been most encouraging. This method can also be used to great advantage in treating advanced carcinoma of the sinuses and nasal passages where the disease is too inaccessible for radium therapy.

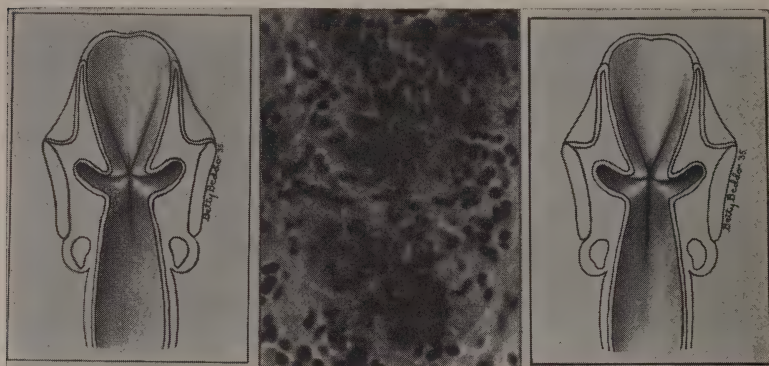


FIG. 6. Early epidermoid carcinoma of the vocal cord treated with deep x-rays by delivering 4,250 r to the sides of the neck during a period of twenty-one days. Two months later after the reaction had disappeared the laryngologist reported only a slight scarring which the artist failed to indicate in the sketch on the right.

After observing the cases treated by Merritt in Washington, D. C., with 220 kv. peak, a filter of 2 mm. copper and 1 mm. aluminum, 20 ma., and a target skin distance of 50 cm., we have installed a similar set of apparatus and have adopted his technic. Of the many modifications of the original Coutard plan, this one impresses us as the most efficient at the present time.

The reader is no doubt thoroughly impressed with the fact that deep therapy is now in a state of progressive improvement. The method is a dangerous one, which should be handled only by an experienced radiologist, but if he be very careful and not too conservative he will obtain some surprisingly good results and very few troublesome sequelæ.

## CERVICAL METASTASES

The stumbling block in the path of all those who treat cancer about the head is the care of metastatic cervical lymph nodes. Neither the surgeons nor the radiologists can point with any great pride to the results obtained in this field. For practical purposes all cases may be divided into three classes, namely, those having no palpable lymph nodes; those having palpable nodes that are considered operable, and those having inoperable metastases in the neck.

The first group usually occasions the greatest controversy because many of our surgical confreres feel that a block dissection should always be done. In cancer of the lip we are opposed to such surgery as a routine procedure, because we are convinced by our own statistics as well as those of Duffy<sup>16</sup> and by the small number of malignant glands found by pathologists in the resected tissue, that the operation is unnecessary in most of the cases on which it is done. At the time of treatment the importance of the appearance of lymph nodes in the neck must be impressed on the patient, and he should be examined at regular intervals for a year or more. In this way the few who develop metastases may be submitted to operation at a favorable stage. The intraoral tumors metastasize much earlier than do those on the lip, and block dissection may be advised at the time that the primary lesion is treated. However, the patient frequently does better if the operative procedure is delayed several months, and a fair number of our cases have remained well without neck surgery. It is our custom to use heavy x-ray therapy administered in fractional doses over the neck as well as implanted long weak radium needles, but their value is not easily estimated.

When operable cervical glands are present at the time that the primary lesion is treated the operation should be done. Unfortunately all surgeons do not establish operability in the same manner, and radiologists see many patients with intractable malignant infiltration in the neck following ill advised surgery. Quick's<sup>17</sup> criteria are based on an ample experience and form a reliable guide. He states:

"Unilateral neck dissections under local anesthesia are done in cases of adult type epidermoid carcinoma presenting clinically involved unilateral nodes with intact capsules and in the presence of a primary growth either controlled already or favorable from that standpoint. Bilateral involvement of nodes is considered an indication of inoperability. Perforation of the node capsule is an indication of inoperability. \* \* \* We regard the metastatic nodes of transitional cell carcinomas, in fact all cellular epidermoids, as being more amenable to radiation therapy than dissection."





FIG. 7. Diffuse undifferentiated infiltrating carcinoma forming a mass three inches in diameter below the ear and extending around the back of the neck to the midline. No primary could be found. Weak radium needles were implanted for seven days as shown in the radiograph. The lower photograph was made a year later when no palpable evidence of carcinoma could be made out.

In inoperable cases having nodes of moderate dimensions Quick inserts radon seeds into the individual nodes after exposing them at operation. The small platinum radium needles may be used in a similar manner. When the masses in the neck reach a considerable size satisfactory regression can frequently be obtained by inserting parallel rows of long platinum radium needles in the base of each tumor at 1 cm. intervals for a period of seven or eight days. This procedure can be safely augmented by doses of deep x-rays totaling 1,800 to 2,000 r and given at the rate of 300 r per day. The more embryonic



tumors of the type shown in Fig. 7 show an excellent response to this form of therapy, and even those containing more adult cells are reduced in size and may remain quiescent for many months. These patients frequently show a rapid improvement and may obtain complete relief of pain for long periods.

#### SUMMARY

1. Many primary epidermoid carcinomas of the lip, mouth, pharynx, larynx, eye and ear can now be completely eradicated with radiation alone.

2. Although rather severe temporary reactions frequently are produced at the time of treatment, the excellent cosmetic results finally obtained make them justifiable.

3. Cervical metastases are best handled by a judicious selection of the surgical or radiation technic best suited to the individual case.

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## OFFICERS

1935-1936

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# AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

## MINUTES OF THE BUSINESS MEETING

The forty-first Annual Meeting of the American Laryngological, Rhinological and Otolological Society, Inc., was held at the Royal York Hotel, Toronto, Ont., on June 3, 4 and 5, 1935.

The business meeting was presided over by the President, Dr. Perry E. Goldsmith, Toronto, Ont. Owing to the temporary absence of President Goldsmith, the first business meeting on the morning of June 3, 1935, was called to order by Chairman Burt R. Shurly, Past-President of the Society.

### *Chairman Shurly:*

At the President's request, I hereby declare the business meeting open, the first procedure being the election of Fellows to Emeritus and Active Fellowship.

### *The Election of Fellows:*

The following Active Fellows of the Society, upon recommendation of the Council, were elected as Emeritus Fellows:

GEORGE E. SHAMBAUGH, M.D., Chicago, Ill.  
HENRY L. WAGNER, M.D., San Francisco, Calif.  
WILLIAM C. BANE, M.D., Denver, Colo.

The following applicants for membership, having fulfilled all of the requirements for election as Active Fellows, were recommended by the Council and each one, being voted upon individually, was elected as an Active Fellow of the Society:

HUGH G. BEATTY, M.D., Columbus, Ohio.  
L. BENNO BERNHEIMER, M.D., Chicago, Ill.  
WALDRON A. CASSIDY, M.D., Omaha, Neb.  
CHARLES E. CONNOR, M.D., St. Paul, Minn.  
SAMUEL J. CROWE, M.D., Baltimore, Md.  
FRED W. GRAEF, M.D., New York, N. Y.  
M. REESE GUTTMAN, M.D., Chicago, Ill.  
VERLING K. HART, M.D., Charlotte, N. C.  
HAROLD L. KEARNEY, M.D., New Orleans, La.  
WILLIAM G. KENNON, M.D., Nashville, Tenn.  
GEORGE H. LANG, M.D., Savannah, Ga.  
WILLIAM L. McDOUGALL, M.D., Atlanta, Ga.

JOHN G. McLAURIN, M.D., Dallas, Tex.  
WILLIAM A. McNICHOLS, M.D., Dixon, Ill.  
PHILIP E. MELTZER, M.D., Boston, Mass.  
ROBERT R. MONTGOMERY, M.D., Long Beach, Calif.  
ARTHUR PALMER, M.D., New York, N. Y.  
FRANKLIN P. SCHUSTER, M.D., El Paso, Tex.  
STEPHAN A. SCHUSTER, M.D., El Paso, Tex.  
J. MORRISSET SMITH, M.D., New York, N. Y.  
PHILIP S. STOUT, M.D., Philadelphia, Pa.  
HARRY K. TEBBUTT, M.D., Albany, N. Y.  
EMANUEL U. WALLERSTEIN, M.D., Richmond, Va.  
EARL LEROY WOOD, M.D., Newark, N. J.

*Appointment of the Auditing Committee:*

The president appointed the following Auditing Committee to audit the accounts of the Treasurer, its report to be presented at the meeting of the Society on the following day:

JOHN J. SHEA.  
E. M. SEYDELL.  
F. R. SPENCER, Chairman.

*Election of the Nominating Committee:*

It having been moved, seconded and carried that the By-Laws be suspended, the President was empowered to name a Nominating Committee. He accordingly named the following as members of this committee:

JOHN F. BARNHILL, Indianapolis, Ind.  
DUNBAR ROY, Atlanta, Ga.  
BURT R. SHURLY, Detroit, Mich.  
EDWARD B. SEWELL, San Francisco, Calif.  
JOSEPH C. PECK, Chicago, Ill.

The Committee was instructed to name its own chairman.

REPORTS OF THE SECTION CHAIRMEN

*Eastern Section.* Frederick T. Hill, Chairman.

The meeting was held in Portland, Me., January 4, 1935. Attendance, 15 percent of the membership, non-members twenty-nine. The President of the Society was present and delivered the first address. Four other papers were presented in the morning and following the business meeting, four others in the afternoon. Dr. C. Stewart Nash was elected as Section Secretary and Dr. Gordon Berry re-elected as a member of the Advisory Membership Committee.

*Southern Section.* Beverly R. Kennon, Chairman.

The meeting was held in Norfolk, Va., January 19, 1935. Eleven papers were presented. Forty-seven percent of the membership of the Section were present together with thirty-three non-members. Dr. E. G. Gill was elected Section Secretary and Dr. Fletcher D. Woodward as a member of the Advisory Membership Committee.

*Middle Section.* Daniel W. Layman, Chairman.

This meeting was held in Indianapolis, Ind., January 23, 1935. Nine papers and demonstrations were presented, one of them by the President of the Society. The attendance consisted of 40 percent of the membership of the section and forty-three non-members. Dr. Walter Theobald was re-elected Secretary of the Section and Dr. B. R. Shurly was elected as a member of the Advisory Membership Committee.

At the business meeting, a memorial meeting was held for Dr. John Carmack and motion made, seconded and carried that a committee composed of Dr. Perry Goldsmith, Dr. William V. Mullin and Dr. Joseph C. Beck present to Mrs. Carmack the resolution read before the memorial meeting together with letters from the Detroit Otolaryngological Society and telegrams received from friends.

*Mid-Western Section.* Edward A. Cary, Chairman.

This meeting was held at Dallas, Tex., January 28 and 29, 1935, under the auspices of the Dallas Academy of Ophthalmology and Otolaryngology. Twenty-four percent of the membership were present together with eighty-two non-members. On the first day six papers were presented and on the second, a symposium on malignant disease was given, consisting of seven parts. On the evening of January 28 the Section was the guest of the Dallas Academy of Ophthalmology and Otolaryngology. Dr. C. C. Cody was elected Section Secretary and Dr. John H. Foster as a member of the Advisory Membership Committee.

*Western Section.* Frank A. Burton, Chairman.

This meeting was held at Los Angeles, Calif., January 18 and 19, 1935. Ten papers were presented during the two sessions. Eighteen percent of the membership of the Section were present, together with thirty-seven non-members. Dr. Fred H. Linthicum was elected Section Secretary and Dr. Sigurd v. Christiernson was elected a member of the Advisory Membership Committee.

*Secretary's Report:*

June 4, 1935.

*Fellows of the Society:*

The fortieth gathering of our organization for the cultivation of social intercourse and the development of scientific work among its members seems to me to be a fit occasion to review our existence a bit, as far as it has to do with our attendance at meetings.

The first meeting was held in New York City under the Presidency of Dr. Edward B. Dench, on April 17, 1896. The organization at that date consisted of 124 members, thirty-three, or 27 percent of whom attended the meeting.

The membership today consists of 436 Fellows, an increase of 350 percent, during our forty years of activity.

The average attendance at annual meetings during this period has been 34 percent of the membership.

The largest attendance was in 1907 in New York City and in 1925 in Atlantic City, with 57 percent of the Society attending each meeting. The smallest meeting was in 1899, in Cincinnati, when 12 percent attended.

Annual meetings have been held in the Eastern Section twenty-nine times, in the Southern, three times, in the Middle, six times, and in the Mid-western and Western Sections, once each.

It is interesting to note that the meetings in Atlantic City, eleven in number, have always drawn the largest percentage of membership.

The average of attendance of the Sections at the annual meetings for the past ten years has been, the Eastern 39 percent, the Southern, 35 percent, the Middle, 40 percent, the Mid-western, 32 percent, and the Western, 18 percent.

As to the interest taken by the Section members in their own meetings, the figures given below will indicate the same enthusiasm for the success of the Society that is shown by the attendance at the annual meetings; the Eastern Section, 24 percent of its membership, the Southern, 30 percent, the Middle, 42 percent, the Mid-western, 34 percent, and the Western, 48 percent of its membership.

So much for the past.

The annual meeting held in Charleston, S. C., under the Presidency of Dr. J. W. Jervey, was a grand success in every way, as those who attended and were privileged to partake of the delightful hospitality of Doctor Jervey's host of friends in Charleston will attest. There just was not time to take it all in.

There were in attendance ninety-five Fellows, 22 percent of the membership. One hundred and seventeen members and guests at-



tended the annual dinner, which took the form of a Southern barbecue.

All but one of the papers scheduled for the scientific meeting were presented.

The Sections were represented as follows: Eastern, 24 percent of its membership; Southern, 40 percent; Middle, 21 percent; Mid-western, 12 percent; and Western, 6 percent.

All of the fourteen candidates recommended by the Council for membership were elected as Active Fellows and all have accepted election. Six Active Fellows of the Society were elected as Emeritus Fellows.

Dr. Edward F. Parker, Charleston, S. C., was nominated by the Society as an Honorary Fellow of the Society and was unanimously elected. This action was confirmed by the Council.

The present membership of the Society consists of:

Active Fellows .....	406
Emeritus Fellows .....	30
Total membership .....	436

There are ten Honorary Fellows.

The membership by Sections consists of:

Eastern .....	211	48 percent of the entire membership
Southern .....	49	11 percent of the entire membership
Middle .....	54	12 percent of the entire membership
Mid-western .....	71	16 percent of the entire membership
Western .....	51	12 percent of the entire membership

Since the last annual meeting, the Society has lost by death, the following members:

*Active Fellows:*

John W. Carmack.....	elected 1929
John F. Dickson.....	elected 1929
Robert C. Howard.....	elected 1930
James J. LaSalle.....	elected 1908
Pinkney V. Mikell.....	elected 1925
William V. Mullin.....	elected 1919
Wendell C. Phillips.....	elected 1895
W. Scott Renner.....	elected 1895
Harmon Smith .....	elected 1903
F. M. Thigpen.....	elected 1895
D. J. Gibb Wishart.....	elected 1900
Edward F. Ziegelman.....	elected 1932

*Honorary Fellow:*

Cornelius G. Coakley.....	elected 1929
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The list of those delinquent in having failed to attend a Section or annual meeting for three consecutive years consists of :

14 members for four years  
4 members for three years

There are in addition forty-five Active Fellows who have not attended a meeting for three years but who are automatically excused under a ruling of the Council that members of twenty years' standing are so excused.

The present list of applications for membership consists of :

Applications approved and these due on November 1 next.....	13
Applications before the Admissions Committee.....	15
Applications on file, not yet acted upon.....	7
<hr/>	
Total active applications.....	35

The attendance of the membership at the various Section meetings held in January last were:

Eastern .....	33	15 percent of its membership
Southern .....	23	25 percent of its membership
Middle .....	21	37 percent of its membership
Mid-western .....	19	20 percent of its membership
Western .....	28	51 percent of its membership

The following were elected by the Sections to serve as members of the various Advisory Membership Committees for a period of three years:

Eastern—G. BERRY.  
Southern—F. D. WOODWARD.  
Middle—B. R. SHURLY.  
Mid-western—J. H. FOSTER.  
Western—S. v. CHRISTIERSON.

In closing my annual report, may I refer to my very keen regret that circumstances over which I have no control prevent my presence at the Toronto meeting which I had looked forward to with so much pleasure of attending.

Our yearly meetings are a wonderful opportunity to study what "fellowship" means and we who are regular in attendance have come to know it to its fullest.

Signed,

ROBERT L. LOUGHRAN,  
*Secretary.*

*Report of the Treasurer:*

Balance on hand, March 19, 1934.....\$ 4,563.54

## RECEIPTS

Sold two Japanese Government Bonds.....	\$ 1,889.67	
Transferred from savings account.....	2,000.00	
1 Dues received for year 1930-31.....	15.00	
4 Dues received for year 1931-32.....	60.00	
11 Dues received for year 1932-33.....	165.00	
55 Dues received for year 1933-34.....	825.00	
298 Dues received for year 1934-35.....	4,470.40	
14 Initiations for year 1934-35.....	140.00	
		<hr/> 9,565.07
Total .....		<hr/> \$14,128.61

## DISBURSEMENTS

## Expenses:

Annual Meeting 1933-34.....	\$ 342.33
Annual Meeting 1934-35.....	125.86
Sectional Meeting .....	199.20
Council Meeting .....	254.84
Publication Department .....	2,977.78
Federal Stamp Tax.....	.42
Treasurer .....	140.00
Secretary .....	528.83

## Purchase:

65 Shares Westinghouse Airbrake.....	6,643.92
25 Shares Jones & Laughlin Pfd.....	
32 Shares American Telephone & Telegraph..	
	<hr/> \$11,213.18

Balance on hand May 27, 1935... \$ 2,915.43

Balance on hand March 19, 1934.....\$ 1,278.18

## RECEIPTS

Interest on savings account.....	\$ 51.47
Dividends on various stocks.....	1,893.51
	<hr/> 1,944.98
Total .....	<hr/> \$ 3,223.16

## DISBURSEMENTS

Purchase of securities.....	\$ 2,000.00
Balance on hand May 27, 1935, savings account No. 21529, Commonwealth Trust Co. of Pittsburgh....	\$ 1,223.16
Total .....	\$ 3,223.16

Statement audited and certified as being correct, stock checked and found to be on hand, Commonwealth Trust Co., Pittsburgh, Pa., W. M. Sheridan, Secretary.

The Treasurer would advise the orderly liquidation of all stock and gradual conversion into recognized securities and the final conversion of these be put into a Trust Fund.

EWING W. DAY,  
*Treasurer.*

*Report of the Auditing Committee:*

Your Auditing Committee has examined the sworn statement of the Commonwealth Trust Company of Pittsburgh, as well as the papers presented to it by the Treasurer and find them correct and in order.

J. J. SHEA,  
E. M. SEYDELL,  
F. F. SPENCER,  
*Committee.*

*Report of the Editor of the Transactions:*

The Transactions for 1934 were completed and ready for distribution on February 1, 1935. Due to a change in printers, it was possible this year to print a smaller number of Transactions, 500, at a saving to the Society of approximately \$1,000 over the cost of the 1933 volume. It is hoped that this saving can be continued for the ensuing year, provided that the size of the volume does not increase.

The editor again bespeaks the coöperation of the Society members in handing in manuscript which at the time of presentation is ready for the printer. This necessitates copy free from any typographical errors, set up in acceptable form for printing. It is absolutely essential that cuts accompany the paper and that all bibliographical references be correct, and properly abbreviated. The cost of cuts prepared by the printer will be charged to the author unless these are furnished through the courtesy of one of the recognized journals in which the article has already appeared. Only through the coöperation of members in the above suggestions can the Transactions



appear at the desired early date and only thereby can the cost of the volume be kept at its present lower figures.

*Editor's Report of Receipts and Disbursements:*

June 1, 1934 to May 27, 1935

Balance on hand June 1, 1934.....\$ 312.97

RECEIPTS

Sale of books and reimbursement of postage...	\$ 61.79	
		61.79
		<hr/>
	\$	374.76

DISBURSEMENTS

Medical Directory .....	\$ 12.00	
Bank stamp .....	2.65	
Printing .....	30.65	
Postage, etc. ....	56.29	
Bank tax and charges.....	5.14	
File supplies .....	8.30	
Editor (1933) .....	100.00	
Editor (1934) .....	100.00	
Reimbursement on book.....	6.00	
		<hr/>
		321.03

May 27, 1935—balance on hand..... \$ 53.73

LYMAN G. RICHARDS,

*Editor.*

*Report of the Nominating Committee:*

Your Nominating Committee presents the following list for the consideration of the Society as officers for the ensuing year:

THOMAS B. CARMODY, Denver, Colo.

HENRY B. ORTON, Newark, N. J.

ROBIN HARRIS, Jackson, Miss.

WILLIAM E. GROVE, Milwaukee, Wis.

HARRY W. LYMAN, St. Louis, Mo.

CARROLL SMITH, Spokane, Wash.

EWING W. DAY, Pittsburgh, Pa.

LYMAN G. RICHARDS, Boston, Mass.

ROBERT L. LOUGHRAN, Sharon, Conn.

JOSEPH C. BECK, Chicago, Ill.

HAROLD I. LILLIE, Rochester, Minn.

HARRINGTON B. GRAHAM, San Francisco, Calif.

J. W. JERVEY, Greenville, S. C.

D. C. JARVIS, Barre, Vt.

W. P. WHERRY, Omaha, Neb.

LEE M. HURD, New York, N. Y.

DON M. CAMPBELL, Detroit, Mich.

PERRY G. GOLDSMITH, Toronto, Ont.

(Signed) THE COMMITTEE,

JOHN F. BARNHILL,

DUNBAR ROY,

BURT R. SHURLY,

EDWARD P. SEWELL,

JOSEPH C. BECK, *Chairman*.

### *Election of the Council:*

The list of candidates for members of the Council rendered at a previous meeting by the Nominating Committee was read and, upon motion, seconded and carried. The Secretary was requested to cast an affirmative vote for the members of the Council as listed. This was done and the members named in the list were declared duly elected.

At a later hour, the newly-elected Council reported that the following officers had been selected to serve for the ensuing year:

*President*—Thomas B. Carmody.

*Vice-Presidents*—

Henry B. Orton, Chairman, Eastern Section.

Robin Harris, Chairman, Southern Section.

William E. Grove, Chairman, Middle Section.

Harry W. Lyman, Chairman, Mid-western Section.

Carroll Smith, Chairman, Western Section.

*Treasurer*—Ewing W. Day.

*Editor*—Lyman G. Richards.

*Secretary*—Robert L. Loughran.

*Council*—

Class A—Joseph C. Beck,

Harold I. Lillie,

Harrington B. Graham.

Class B—J. W. Jervey,

D. C. Jarvis,

W. P. Wherry.

Class C—Lee M. Hurd,

Don M. Campbell,

Perry G. Goldsmith.

### *Report of Representatives of the American Board of Otolaryngology:*

In the absence of Doctor Shurly, Senior Director of the Board, Dr. Ralph A. Fenton reported that 162 candidates had been examined by the Board at four examinations held in Cleveland, Ohio., in Butte,

Mont., in San Antonio, Tex., and in Chicago, Ill. The next meeting of the Board will be held in New York City.

The activities of the Board were summarized in an analysis for 1925 through 1933 which was made to the Advisory Board for the Medical Specialties and submitted as a part of this report. On motion duly made and seconded, this report was accepted.

On motion duly made and seconded, it was voted to send to Doctor Loughran a suitable telegram expressing the Society's regret at his unavoidable absence through illness.

#### *Standardization of Hearing Aids:*

Dr. Edmund P. Fowler brought to the attention of the Society the danger of exploitation of deafened persons by manufacturers of hearing aids. In this connection, he read a letter from Doctor Knudsen, Physicist at the University of California, explaining the importance of financing scientific work leading to the proper evaluation of all hearing aids, thus enabling the physician to prescribe the most suitable aid for the particular patient. Some financial support of proper scientific research work is being considered by the American Otological Society and this Society is asked to share in the expense of this work. Doctor Fowler moved that the Triological Society contribute \$250 towards this object. It was moved, seconded and passed that the matter be referred for consideration by the Council with power to act. In this connection, a resolution already passed by the American Otological Society was submitted by Dr. Gordon Berry as follows:

"Whereas knowledge has come to us that certain distributors and agents of electric hearing devices have offered to physicians and others a bonus or commission for referring to them hard-of-hearing persons to whom they effect a sale, be it resolved by the American Laryngological, Rhinological and Otological Society that it condemns as unethical and unfair to the purchaser the practice on the part of any agent or distributor of a hearing device who offers to pay to any physician a bonus or commission for referring a purchaser of such device. Acceptance of such a bonus or commission by a practicing physician is interpreted as a violation of the principles of ethical practice." It was moved and seconded that this resolution be adopted.

#### *Limitation of Membership:*

After expressing the opinion of several members of the Society, that the Society, owing to its rapid increase in membership, might become in a short time too large and unwieldy, Dr. William B.

Chamberlain offered a motion that the membership in the Society be limited to 500. This was seconded by Doctor Barnhill, and an amendment thereto offered by Dotcor Day that the membership be circularized by postcards in order to secure the opinion of individual members of the Society on this subject and that a report be ready for the Council at the mid-winter meeting. Doctor Hastings then offered, in discussion, the opinion that with the normal decrease due to deaths and transfers to the Emeritus list, the size of the Society would probably normally remain well within bounds. He felt that circularization by a postcard would be of little use, as opinions would be expressed with little or no serious consideration. He favored limitation of the Society to 450 and no more. Doctor Chamberlain then voiced the opinion that the idea of admission to this Society by invitation only should be stressed and that the tendency to admission by application should be strongly repressed. Doctor Day voiced his support for this idea. A vote was then taken as to Doctor Day's amendment that the Society be circularized by a postcard, and on motion duly made and seconded this amendment was defeated. Doctor Chamberlain then moved that the membership be restricted to 450 instead of 500. This motion was seconded by Doctor Barnhill, and was amended by Doctor Mosher, that the number be left to the discretion of the Council. This amendment was seconded. Doctor Chamberlain accepted this amendment, provided that the Council did not raise the number to over 500. On motion duly made and seconded, this motion was carried.

On motion duly made and seconded, it was voted to extend the thanks of the Society to the Toronto Otolaryngological Society and likewise to the Ladies of Toronto and to the Artists and Letters' Club for the delightful entertainment afforded during the Society's visit to Toronto.

The Society than expressed its great appreciation of the honor done it by the presence of its guest, Mr. Patterson of London. Mr. Patterson responded with appropriate remarks.

*Introduction of the newly-elected President:*

The President named Drs. John Barnhill and John Shea as a committee to escort the incoming President to the rostrum. President Carmoy spoke briefly, expressing his great appreciation of the honor done to the Western Laryngologists by his election and expressed the hope that his administration might continue to achieve the high standards set by his predecessors.

Business sessions were held on the mornings of June 3, 4 and 5, 1935.



## NECROLOGY

### DR. JOHN W. CARMACK

Dr. John W. Carmack died on December 5, 1934. He was born and educated in Indiana, graduating from the Indiana University School of Medicine. After graduating he practiced general medicine for a number of years in Indianapolis. At the outbreak of the World War he volunteered in the Otolaryngological Division of the Medical Service, and was stationed at Camp Taylor, near Louisville. It was in this service that he received his first inspiration in otolaryngology.

After the war he established himself in Indianapolis, limiting his practice to diseases of the ear, nose and throat. He took postgraduate work in these branches at the Indiana University Medical Center for a period of five years, and was during that time assigned clinical work as an instructor in the institution. In 1930 he was advanced to the chairmanship of the department of otolaryngology, and would no doubt have been given the professorship in a few years.

Doctor Carmack was elected an active fellow of this society in 1929, and in 1934, at the time of his death, was chairman of the Middle Section.

Doctor Carmack was a tireless worker and made rapid advancement. He was endowed with charming social qualities and made fast friends readily. His death is a distinct loss to the profession both socially and scientifically.

He was married and leaves a wife and one son, John Carmack, Jr., age ten.

### DR. JAMES J. LASALLE

Dr. James J. Lasalle, of Phoenix, Ariz., an active member of this Society since 1908, died on July 22, 1934.

He graduated from the University of Michigan in 1896, studied in Germany during the three succeeding years, and began general practice in Toledo, Ohio, in 1899.

In 1903 he undertook postgraduate work in otolaryngology in Vienna, and returned to Toledo to take up the practice of this specialty, which he pursued uninterruptedly for twenty years. He was a member of the Toledo Medical Society during this time.

In 1930 he moved to Phoenix, Ariz., where he continued to work until ill health in 1932 compelled him to relinquish his practice.

He was the author of numerous medical publications, one of them in collaboration with Prof. Gustav Alexander in Vienna.

**DR. D. J. GIBB WISHART**

In the death in Toronto on June 5, 1934, of Dr. D. J. G. Wishart, this Society lost one of its most distinguished and loyal members, an active member since 1900. He occupied a conspicuous place among the Canadian constituent of our organization, being a member of the Council in 1916, 1917 and 1918.

Doctor Wishart was born at Madoc, Ontario, on September 11, 1865. His early education was acquired in the Brantford Collegiate Institute, from which he matriculated to the University of Toronto in Arts, taking his Bachelor's degree in 1882. He received the degree of Doctor of Medicine from McGill University in 1885, in which year he also became a licentiate of the Royal College of Physicians in London.

He became a member of the American College of Surgeons in 1913, and belonged also to the Royal Society of Medicine in London.

His entire period of practice was spent in Toronto, where he occupied himself continuously as a teacher and practitioner of otolaryngology. He was the chief of this department in the Hospital for Sick Children, from 1900 to 1907; on the staff in otolaryngology in the Toronto General Hospital from 1890 to 1922; and was chief of this department from 1914 to 1922. He occupied numerous teaching positions, holding the professorship of otolaryngology in the University of Toronto from 1903 to 1922.

His memberships in other societies comprised a notable list—Canadian, British and American. During the war he served with distinction at home as consultant in otolaryngology with the rank of lieutenant colonel.

Appreciative testimonials of the Toronto associates of Doctor Wishart clearly indicate the high esteem in which he was held by his confreres, and the eminent position which he occupied in the front ranks of our specialty.

**DR. HARMON SMITH**

On December 11, 1934, this Society lost through the death of Dr. Harmon Smith, one of its most loyal and outstanding members. He was born at McDonough, Ga., graduating from the University of Georgia in 1892.

Following his graduation from Bellevue Hospital Medical College in 1897, he served his internship at King's County Hospital and Loomis Sanatorium.

He was a fellow of the American College of Surgeons, a member of the New York Academy of Medicine, American Medical Association, Medical Society of the State of New York, and the American Laryngological Association, of which he was a past president.

In 1921 he was chairman of the Eastern Section of this Society. In 1922 he was honored by the University of Georgia with the degree of Doctor of Science.

Doctor Smith was chief surgeon of the Manhattan Eye, Ear, Nose and Throat Hospital, consulting laryngologist to the Memorial Hospital, to the Babies' Hospital, and the Monmouth Memorial Hospital of Long Branch, N. J.

He was a collaborating author of one of the outstanding textbooks of diseases of the ear, nose and throat.

Through the death of Doctor Smith the profession is deprived of a distinguished practitioner, and this Society of a devoted and enthusiastic member.

#### **DR. PINCKNEY VENNING MIKELL**

Dr. Pinckney Venning Mikell was born on Edisto Island, S. C. He received an A.B. degree from the University of South Carolina, and his medical degree from the Medical College of South Carolina at Charleston, in 1900. He settled in Columbia and was engaged in general practice of medicine for several years, after which he specialized in the diseases of the eye, ear, nose and throat. During his thirty-four years' practice in Columbia, Doctor Mikell was continuously a progressive and outstanding leader, not only in medical affairs, but in any civic organization that worked for the betterment of his fellow man.

As a physician he ranked as a leader. Here again his work was not confined to his specialty alone, for he was always actively interested in everything that tended to enhance the practice of medicine in all lines. He was a member of the Columbia Medical Society for over thirty years. During this time he presented many scientific papers, served on important committees, and was honored with the presidency. For several years he served as chief of staff at the Columbia Hospital.

He was a member of the American Medical Association, the American College of Surgeons, and a licentiate of the American Board of Otolaryngology, and attended frequently the national meetings of his specialty, contributing case reports and papers.

He became a member of this Society in 1925.

Doctor Mikell possessed to a rare degree the charm and idealism of the old school even in the practice of a modern specialty. Endowed

with a vigorous personality, the expectation of many years of health and medical productiveness was cut short by his accidental death on July 31, 1934.

#### **DR. W. SCOTT RENNER**

Doctor Renner, member of our Society for forty years, died at his home in Buffalo after months of failing health. Doctor Renner was for many years active in our Society and had a large share of influence in the work of our specialty in Buffalo. He was professor of otolaryngology in the University of Buffalo for a long period of time.

#### **DR. ROBERT C. HOWARD**

Dr. Robert C. Howard, elected active fellow of this Society in 1930, died on May 4, 1935, was born in Frederick, Md., on August 16, 1883. He was graduated from the College of Pharmacy, Columbia University, in 1903, and from Cornell Medical School in 1911. Following his internship at New York Hospital in 1913, he pursued postgraduate work at Memorial Hospital in 1914.

He occupied the position of instructor in otolaryngology at Cornell Medical School for the succeeding ten years, being at the same time an instructor in this specialty at the Manhattan Eye, Ear, Nose and Throat Hospital. He was also clinical professor in the department of otology at the Polyclinic hospital and Medical School.

Doctor Howard was a member of the New York County Medical Society, New York Academy of Medicine, American College of Surgeons, and the New York Medical Union. He died of a heart attack while working about his summer home at Greenwood Lake, N. J.

#### **DR. JOHN F. DICKSON**

Dr. John F. Dickson, a member of this Society since 1929, died on April 5, 1934. He was born in Renfrew, Canada, in 1855, but early in his life removed to Portland, Ore., where he was in active practice for over forty years. He was graduated from Toronto University, and did postgraduate work at Edinburgh, London and Vienna.

Doctor Dickson was a member of the American Medical Association, a fellow of the American College of Surgeons, and he was one of the founders of the Pacific Coast Oto-ophthalmological Society, a Past President of the Oregon State Medical Society, and emeritus professor of ophthalmology in the University of Oregon Medical School.



**DR. EDWARD F. ZIEGELMAN**

Dr. Edward F. Ziegelman, a member of this Society since 1932, died suddenly on October 12, 1934, while on his way to a meeting of the American College of Surgeons in Boston, Mass.

Doctor Ziegelman was graduated from Jefferson Medical College, of Philadelphia, in 1912. At the outset of his practice he was associated with Dr. Thomas M. Joyce, of Portland, Ore., with whom he gained experience, particularly in surgery of the head and neck, and with whom he served overseas in Base Hospital No. 46. Later, becoming especially interested in these regions, he determined to devote himself exclusively to the practice of otolaryngology, first as instructor in the University of Oregon Medical School, later joining the staff of this department in Stanford University, San Francisco, where he soon demonstrated his great enthusiasm for anatomical investigation.

He traveled widely both in this country and abroad. Many of his anatomical contributions were characterized by originality and great thoroughness, and remain as a memorial to his untiring energy and enthusiasm.

Doctor Ziegelman was a member of the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, the Pacific Coast Oto-ophthalmological Society, and the American College of Surgeons.

In his death the Society has lost a member whose contributions had already given promise of a brilliant future.

**DR. WILLIAM V. MULLIN**

Dr. William V. Mullin died in Cleveland on April 25, 1935. Dr. Mullin was born in Iowa City, Iowa, on February 14, 1884. After preliminary education in Iowa City, he attended the University of Iowa from 1902 to 1904. He received his Doctor's degree at the University of Denver in 1908.

He completed his internship at St. Joseph's Hospital in Denver, and in 1909 undertook general practice at Holly, Colo., finally establishing himself definitely at Colorado Springs the same year. He began to specialize in Otolaryngology in 1910.

In 1913 he was married to Miss Louise M. Nichols, of Colorado Springs, and in 1914 he did postgraduate work in otolaryngology at the Killian and Halle Clinics in Germany. When the United States entered the World War, Doctor Mullin became a lieutenant in the Medical Corps and was stationed at General Hospital No. 19, at Oteen, N. C., throughout the conflict.

After the war he returned once more to Colorado Springs and remained there until 1926, when he was appointed the head of the otolaryngological department of the Cleveland Clinic.

He was a frequent contributor to medical publications, and an influential member of many medical societies. Among them were the American Medical Association, in which he was first secretary, and then, in 1921, chairman of the Section of Otology, Rhinology and Laryngology. He was a member of the Ohio State Medical Association, in which he filled a similar office in 1933, and of the American Laryngological Association, of which he was vice-president in 1926, treasurer in 1933, and secretary in 1934. He was also a member of the American Otological Society. Doctor Mullin moreover was an examiner on the American Board of Otolaryngology, fellow of the American College of Surgeons, and a member of the American Academy of Ophthalmology and Otolaryngology.

He became a member of this Society in 1919, being chairman of the Mid-western Section in 1921, and a member of the Council in 1924, 1925 and 1926.

At the time of his death Doctor Mullin was chief of the department of otolaryngology at the Cleveland Clinic, at Cleveland, and he was known throughout the country as a practitioner and teacher of the highest ability, and was beloved by all his associates.

#### **DR. WENDELL C. PHILLIPS**

Dr. Wendell C. Phillips, former president of the American Medical Association, and a charter member of this Society, died in New York on November 16, 1934. Doctor Phillips was born in St. Lawrence County, New York, and received his medical training in this city. He was graduated from the medical department of the University of the City of New York in 1882 and began practice here soon after. In addition to his connection with several hospitals in this city, Doctor Phillips was associated with many scientific associations, including New York Academy of Medicine, American Otological Society, and the American College of Surgeons.

He was at one time professor of otology at the New York Post-graduate Medical School, and oral surgeon and director of the Manhattan Eye, Ear, Nose and Throat Hospital. He was president of the Medical Society of the State of New York in 1912. Doctor Phillips was honored by election to the presidency of the American Medical Association, the first otolaryngologist to fill this important position.

He was the author of a textbook on diseases of the ear, nose and throat, and of many articles and monographs pertaining to otology and laryngology.

It was in his relationship to this Society, however, that Doctor Phillips most fully found expression for the affection which he felt for his colleagues in the specialty. Since joining the Trilogical Society in 1895 he had never missed attendance at an annual meeting, over a period of forty years. He was the Society's secretary from 1901 to 1906, and its president in 1907. He was a member of the Council on no less than four separate occasions.

Great as were his other distinctions, Doctor Phillips will be longest remembered by many for the pioneer work he did in the founding of the American Federation of Organizations for the Hard of Hearing. Through his efforts the accomplishments of this body have spread throughout the length and breadth of the land to the aid and comfort of millions of individuals handicapped by deafness.

Through the inspiring example of Wendell Phillips this work is widening its scope. It has become his chosen memorial.

#### DR. C. G. COAKLEY

Dr. C. G. Coakley, professor of laryngology and otology at Columbia University since 1914, died on November 22, 1934. Doctor Coakley was born in Brooklyn, received his A.B. degree from the College of the City of New York in 1884, and his M.A. degree three years later.

In 1887 he was graduated from Cornell University and Bellevue Medical School, receiving first honors. He served his internship at Bellevue Hospital in 1887, and then joined the faculty of the New York University Medical College, first as a lecturer on anatomy and later as instructor in histology.

Two years later he returned to New York University as clinical professor of laryngology and in 1905 was made professor in that subject. In 1914 he went to Columbia University, where he had since held the position of professor and head of the department.

He was elected a member of this Society in 1900 and was made an honorary fellow in 1929. He was a member of the American Otological Society and the American Laryngological Association, of which he was at one time president.

He was the author of a volume entitled "Diseases of the Nose and Throat," published in 1899 and since printed in five editions and used widely as a medical textbook.





## ACTIVE FELLOWS

\*Has attended a section or annual meeting during the year

- \*1913 Abbott, William J....10515 Carnegie Ave., Cleveland, Ohio
- 1901 Abraham, Joseph H.....130 W. 58th St., New York, N. Y.
- 1924 Adams, Frank M.....122 Waterman St., Providence, R. I.
- \*1925 Alden, Arthur M.....Frisco Bldg., St. Louis, Mo.
- \*1913 Alexander, Lawrence D....30 W. 59th St., New York, N. Y.
- \*1908 Allen, John H.....32 Deering St., Portland, Me.
- 1932 Anderson, Carl M.....Mayo Clinic, Rochester, Minn.
- \*1924 Arbuckle, Millard F...3720 Washington Blvd., St. Louis, Mo.
- 1904 Ard, Frank C.....Lake Wales, Fla.
- \*1933 Ashley, Rae E.....384 Post St., San Francisco, Cal.
- 1899 Atkinson, J. William.....485 Maple Ave., Glen Rock, N. J.
- \*1905 Babbitt, James A.....1912 Spruce St., Philadelphia, Pa.
- 1925 Babcock, James W.....20 E. 53rd St., New York, N. Y.
- \*1926 Ballenger, Howard C....25 E. Washington St., Chicago, Ill.
- \*1922 Barlow, Roy A.....Medical Arts Bldg., Rochester, N. Y.
- 1915 Barndollar, William P..Westinghouse Bldg., Pittsburgh, Pa.
- 1922 Baum, Harry L.....Republic Bldg., Denver, Colo.
- 1935 Beatty, Hugh G.....681 E. Broad St., Columbus, Ohio
- \*1903 Beck, Joseph C.....185 N. Wabash Ave., Chicago, Ill.
- 1935 Bernheimer, L. Benno...104 S. Michigan Ave., Chicago, Ill.
- \*1922 Berry, Gordon.....Medical Arts Bldg., Worcester, Mass.
- \*1905 Birkett, Herbert S....1190 Mountain St., Montreal, Canada
- \*1926 Bishop, Vernon L....Medical Arts Bldg., Rochester, N. Y.
- \*1934 Blackmar, Francis B.....Swift Kyle Bldg., Columbus, Ga.
- \*1912 Blackwell, Hugh B.....8 E. 54th St., New York, N. Y.
- \*1928 Blassingame, Chas. D.....P. & S. Bldg., Memphis, Tenn.
- 1928 Bowers, Chester H....1136 W. Sixth St., Los Angeles, Cal.
- 1918 Bowers, Wesley C.....17 E. 61st St., New York, N. Y.
- 1898 Braislin, William C.....425 Clinton Ave., Brooklyn, N. Y.
- \*1915 Braun, Alfred.....1000 Park Ave., New York, N. Y.
- 1932 Bristow, Walter J.....Medical Bldg., Columbia, S. C.
- 1931 Brooks, Earl B.....Stuart Bldg., Lincoln, Neb.
- \*1915 Brown, Clayton M.....510 Delaware Ave., Buffalo, N. Y.
- 1911 Brown, H. Beattie....115 Park Ave., Saranac Lake, N. Y.
- 1909 Brown, John E.....370 E. Town St., Columbus, Ohio
- 1916 Brown, J. Mackenzie...1136 W. Sixth St., Los Angeles, Cal.

- 1922 Brown, Louis E.....2nd National Bldg., Akron, Ohio
- \*1923 Brown, Morrow D.....Republic Bldg., Denver, Colo.
- 1917 Brumm, Seth A.....Medical Arts Bldg., Philadelphia, Pa.
- 1903 Bryant, William Sohler....30 E. 40th St., New York, N. Y.
- \*1923 Buckley, Robert E.....50 E. 63rd St., New York, N. Y.
- 1915 Burns, Louis J.....1930 Chestnut St., Philadelphia, Pa.
- 1922 Burton, Frank A.....Bank of America, San Diego, Cal.
- \*1914 Butler, Ralph.....1930 Chestnut St., Philadelphia, Pa.
- 1923 Callison, James G.....50 E. Tenth St., New York, N. Y.
- \*1915 Campbell, Don M.....David Whitney Bldg., Detroit, Mich.
- \*1929 Campbell, Edward H.....314 Kent Rd., Cynwyd, Pa.
- \*1912 Carmody, Thomas E.....Metropolitan Bldg., Denver, Colo.
- 1915 Carter, William W.....140 E. 54th St., New York, N. Y.
- \*1932 Cary, Edward H.....Medical Arts Bldg., Dallas, Texas
- 1935 Cassidy, Waldren A.....Medical Arts Bldg., Omaha, Neb.
- 1927 Chamberlain, Charles T.....193 11th St., Portland, Ore.
- \*1908 Chamberlin, William B...Carnegie Med. Bldg., Cleveland, O.
- \*1922 Charlton, C. Coulter.....124 S. Ill. Ave., Atlantic City, N. J.
- 1900 Chenery, William E...377 Commonwealth Ave., Boston, Mass.
- 1933 Christierson, Sigurd v.....490 Post St., San Francisco, Cal.
- \*1924 Clerf, Louis H.....128 S. Tenth St., Philadelphia, Pa.
- \*1907 Coates, George M.....1721 Pine St., Philadelphia, Pa.
- 1922 Cobb, Edwin.....Marshalltown, Iowa
- \*1932 Cody, Claude C., Jr.....1304 Walker Ave., Houston, Texas
- \*1914 Cohen, Lee.....1820 Eutaw Pl., Baltimore, Md.
- \*1920 Colver, Benton N.....304 N. Boyle Ave., Glendale, Cal.
- \*1935 Connor, Charles E.....408 St. Peter St., St. Paul, Minn.
- 1916 Cooper, Claude E.....Metropolitan Bldg., Denver, Colo.
- 1933 Costen, James B.....Beaumont Bldg., St. Louis, Mo.
- 1912 Cox, Gerard H.....Strathaven Apts., Glen Cove, N. Y.
- 1906 Craig, Robert H...510 Sherbrooke St., West, Montreal, Can.
- 1926 Crane, Claude G.....353 Washington Ave., Brooklyn, N. Y.
- 1935 Crowe, Samuel J.....4332 N. Charles St., Baltimore, Md.
- 1895 Culbert, William L.....140 E. 54th St., New York, N. Y.
- 1904 Culp, John F.....224 Pine St., Harrisburg, Pa.
- \*1934 Cummings, George O.....47 Deering St., Portland, Me.
- 1928 Cuning, Daniel S.....145 E. 54th St., New York, N. Y.
- \*1916 Cutler, Franklin E.....Hanna Bldg., Cleveland, O.
- 1930 Dabney, Virginius....1633 Conn. Ave., Washington, D. C.
- 1931 Darrow, C. Howard....Metropolitan Bldg., Denver, Colo.
- 1931 Davis, Ralph F.....Medical Arts Bldg., Portland, Ore.
- \*1895 Day, Ewing W....616 Commercial St., Provincetown, Mass.

- \*1930 Day, Kenneth M.....121 University Pl., Pittsburgh, Pa.
- \*1914 Dean, Lee Wallace.....Washington Univ., St. Louis, Mo.
- 1895 Dench, Edward B.....40 W. 51st St., New York, N. Y.
- 1918 Dennis, Frank L.....342 W. Laurel St., San Diego, Cal.
- \*1931 Depping, Charles W.....17 W. 54th St., New York, N. Y.
- \*1922 Dickie, J. K. Milne.....Medical Arts Bldg., Ottawa, Canada
- 1934 Dintenfass, Henry.....1305 Spruce St., Philadelphia, Pa.
- \*1932 Dixon, O. Jason.....Professional Bldg., Kansas City, Mo.
- 1924 Dougherty, Daniel S.....111 W. 85th St., New York, N. Y.
- 1923 Douglass, Beaman.....143 W. 57th St., New York, N. Y.
- 1932 Dowling, Jay T.....1101 Terry Ave., Seattle, Wash.
- 1926 Drury, Dana W.....483 Beacon St., Boston, Mass.
- 1898 Duel, Arthur B.....135 E. 64th St., New York, N. Y.
- \*1920 Dunning, Henry S.....33 E. 68th St., New York, N. Y.
- 1916 Durkee, John W.....34 Eighth Ave., Brooklyn, N. Y.
- 1921 Dworetzky, Julius P.....7 Law St., Liberty, N. Y.
- \*1915 Dwyer, James G.....300 Park Ave., New York, N. Y.
- 1900 Eagleton, Wells P.....15 Lombardy St., Newark, N. J.
- \*1928 Equen, Murdock S.....Medical Arts Bldg., Atlanta, Ga.
- \*1924 Ersner, Matthew S.....1915 Spruce St., Philadelphia, Pa.
- \*1914 Eves, Curtis C.....1910 Spruce St., Philadelphia, Pa.
- \*1914 Fairbairn, John F.....925 Delaware Ave., Buffalo, N. Y.
- 1900 Farrell, Thomas H.....236 Genessee St., Utica, N. Y.
- \*1934 Faulkner, E. Ross.....101 E. 58th St., New York, N. Y.
- 1921 Faunce, Calvin B.....412 Beacon St., Boston, Mass.
- \*1923 Fenton, Ralph A.....Medical Arts Bldg., Portland, Ore.
- 1926 Figi, Frederick A....1013 First St., S.W., Rochester, Minn.
- \*1930 Fisher, James A.....Jersey Cent. Bldg., Asbury Park, N. J.
- \*1915 Fisher, Stanwood E.....723 Congress St., Portland, Me.
- 1899 Fleming, Ernest W.....523 W. Sixth St., Los Angeles, Cal.
- 1927 Fletcher, Harold A.....490 Post St., San Francisco, Cal.
- \*1915 Forbes, Henry H.....40 E. 61st St., New York, N. Y.
- 1911 Forsyth, Edgar A.....645 W. Ferry St., Buffalo, N. Y.
- \*1932 Foster, John H.....1304 Walker Ave., Houston, Texas
- 1907 Foster, John M.....Metropolitan Bldg., Denver, Colo.
- \*1912 Fowler, Edmund P.....140 E. 54th St., New York, N. Y.
- 1926 Fowler, Robert H.....165 E. 64th St., New York, N. Y.
- 1916 Frank, Ira.....104 S. Michigan Blvd., Chicago, Ill.
- 1915 Friesner, Isidore.....36 E. 73rd St., New York, N. Y.
- \*1921 Fuller, Theron E.....National Bank Bldg., Texarkana, Ark.
- \*1924 Furstenberg, Albert C.....Univ. Hsp., Ann Arbor, Mich.
- 1900 Gallaher, Thomas J.....California Bldg., Denver, Colo.

- 1931 Galloway, Thomas C.....636 Church St., Evanston, Ill.
- \*1924 Gill, Elbyrne G.....711 S. Jefferson St., Roanoke, Va.
- \*1934 Gill, William D....Medical Arts Bldg., San Antonio, Texas
- \*1922 Gittens, T. R.....Davidson Bldg., Sioux City, Ia.
- \*1904 Goldsmith, Perry G....Medical Arts Bldg., Toronto, Canada
- \*1899 Goldstein, Max A.....912 S. Kingshighway, St. Louis, Mo.
- \*1926 Gorman, James R.....725 Church St., Lynchburg, Va.
- 1935 Graef, Fred W.....80 E. 77th St., New York, N. Y.
- \*1915 Graham, Harrington B....490 Post St., San Francisco, Cal.
- 1904 Greene, D. Crosby, Jr..85 Dudley Rd., Newton Centre, Mass.
- \*1919 Greene, James S.....126 E. 39th St., New York, N. Y.
- \*1913 Greene, Joseph B.....Haywood Bldg., Asheville, N. C.
- 1921 Gregg, John B....2nd National Bk. Bldg., Sioux Falls, S. D.
- \*1928 Grove, William E...324 E. Wisconsin Ave., Milwaukee, Wis.
- 1916 Guggenheim, Louis K.....Carleton Bldg., St. Louis, Mo.
- 1924 Guttman, John.....1235 Park Ave., New York, N. Y.
- 1935 Guttman, M. Reese.....185 N. Wabash Ave., Chicago, Ill.
- \*1931 Hagens, Elmer W.....30 N. Michigan Ave., Chicago, Ill.
- \*1895 Halsted, Thomas H.....Chimes Bldg., Syracuse, N. Y.
- 1930 Hampton, Robert R....Boston Bldg., Salt Lake City, Utah
- 1928 Hansel, French K.....634 N. Grand Ave., St. Louis, Mo.
- \*1922 Harkness, Gordon F.....Putnam Bldg., Davenport, Ia.
- \*1931 Harrell, Voss.....Hofman Bldg., Detroit, Mich.
- \*1930 Harris, Robin.....Lamar Bldg., Jackson, Miss.
- 1900 Harris, Thomas J.....104 E. 40th St., New York, N. Y.
- 1935 Hart, Verling K.....106 W. Seventh St., Charlotte, N. C.
- \*1906 Hastings, Hill.....1136 W. Sixth St., Los Angeles, Cal.
- 1927 Hasty, Frederick E....Medical Arts Bldg., Nashville, Tenn.
- \*1926 Hayden, Austin A.....25 E. Washington St., Chicago, Ill.
- \*1915 Hays, Harold.....133 E. 58th St., New York, N. Y.
- \*1906 Hedges, Halsted S....104 E. Market St., Charlottesville, Va.
- 1920 Heller, Isaac M.....115 E. 61st St., New York, N. Y.
- 1926 Hempstead, Bert E.....Mayo Clinic, Rochester, Minn.
- 1931 Higbee, David R.....3245 Fourth St., San Diego, Cal.
- \*1920 Hill, Fred T.....Professional Bldg., Waterville, Me.
- 1913 Horn, John.....104 E. 40th St., New York, N. Y.
- \*1932 Houser, Karl M.....1826 Pine St., Philadelphia, Pa.
- 1916 Howell, Hampton P.....140 E. 54th St., New York, N. Y.
- \*1912 Hubby, Lester M.....620 Park Ave., New York, N. Y.
- \*1924 Hubert, Louis.....161 E. 79th St., New York, N. Y.
- \*1926 Hunt, Wesley M.....33 E. 68th St., New York, N. Y.
- \*1901 Hurd, Lee M.....39 E. 50th St., New York, N. Y.



- \*1932 Husik, David M.....1930 Chestnut St., Philadelphia, Pa.
- \*1909 Iglauer, Samuel.....707 Race St., Cincinnati, Ohio
- 1916 Imperatori, Charles J.....108 E. 38th St., New York, N. Y.
- 1925 Ingersoll, Edwin S.....26 S. Goodman St., Rochester, N. Y.
- 1920 Inglis, H. J.....43 Bay State Rd., Boston, Mass.
- \*1924 Israel, Sidney.....Niels Esperson Bldg., Houston, Texas
- \*1901 Jackson, Chevalier.....3432 N. Broad St., Philadelphia, Pa.
- 1899 Jackson, Oliver H.....200 Elm St., Fall River, Mass.
- \*1922 Jarvis, D. C.....Quarry Bank Bldg., Barre, Vt.
- \*1930 Jenkins, William H.....1738 M. St., Washington, D. C.
- \*1909 Jervey, J. W.....101 Church St., Greenville, S. C.
- 1928 Jesberg, Simon.....500 S. Lucas Ave., Los Angeles, Cal.
- \*1933 Johnson, Henry P.....16 Sheffield St., Portland, Me.
- 1926 Johnston, William H...1421 Chapala St., Santa Barbara, Cal.
- 1925 Jones, Arthur C.....Eastman Bldg., Boise, Idaho
- \*1919 Jones, Charles C.....19 Garfield Pl., Cincinnati, Ohio
- \*1906 Jones, Clarence P.....3117 West Ave., Newport News, Va.
- \*1931 Jones, David H.....140 E. 54th St., New York, N. Y.
- 1918 Jones, Isaac H.....1930 Wilshire Blvd., Los Angeles, Cal.
- 1928 Jones, Marvin F.....121 E. 60th St., New York, N. Y.
- 1935 Kearney, Harold L.....3439 Prytania St., New Orleans, La.
- 1921 Keeler, J. Clarence....Medical Arts Bldg., Philadelphia, Pa.
- 1924 Kelley, Isaac D., Jr.....Beaumont Bldg., St. Louis, Mo.
- \*1928 Kelly, Joseph D.:.....140 E. 54th St., New York, N. Y.
- \*1904 Kennon, Beverly R.....Medical Arts Bldg., Norfolk, Vt.
- 1935 Kennon, William G.....708 Church St., Nashville, Tenn.
- \*1919 Kernan, John D.....120 E. 75th St., New York, N. Y.
- 1926 Kiehle, Frederick A.....Medical Arts Bldg., Portland, Ore.
- 1924 King, Edward.....Carew Tower Bldg., Cincinnati, Ohio
- \*1924 Kistner, Frank B.....Mayer Bldg., Portland, Ore.
- \*1910 Kittredge, Frank E.....Masonic Temple, Nashua, N. H.
- \*1933 Kline, Oram J.....414 Cooper St., Camden, N. J.
- 1926 Knisley, Alan D.....Lima Trust Bldg., Lima, Ohio
- 1896 Knowles, William F.....Lenox, Mass.
- 1907 Kopetzky, Samuel J.....51 W. 73rd St., New York, N. Y.
- 1934 Kuhn, Hugh A.....5231 Hohman St., Hammond, Ind.
- \*1934 Kully, Barney M.....Medical Arts Bldg., Omaha, Neb.
- 1935 Lang, George H.....202 Liberty St., E., Savannah, Ga.
- \*1924 La Rue, Claude L....The Highland Clinic, Shreveport, La.
- \*1930 Law, Frederick M.....140 E. 54th St., New York, N. Y.
- \*1915 Layman, Daniel W....Medical Arts Bldg., Indianapolis, Ind.
- 1896 Lederman, M. D.....106 E. 84th St., New York, N. Y.

- \*1933 LeJeune, Francis E...632 Maison Blanch, New Orleans, La.
- 1923 Lemere, Henry B...6381 Hollywood Blvd., Los Angeles, Cal.
- 1922 Levy, Louis.....Bank of Commerce Bldg., Memphis, Tenn.
- 1913 Lewis, Eugene R.....Roosevelt Bldg., Los Angeles, Cal.
- 1915 Lewis, Fielding O.....259 S. 17th St., Philadelphia, Pa.
- \*1923 Lewy, Alfred.....25 E. Washington St., Chicago, Ill.
- \*1929 Lierle, Dean M.....University Hsp., Iowa City, Iowa
- 1921 Lillie, Harold I.....Mayo Clinic, Rochester, Minn.
- 1930 Linthicum, Frederick H...Pac. Mut. Bldg., Los Angeles, Cal.
- \*1923 Looper, Edward A....104 W. Madison St., Baltimore, Md.
- \*1932 Loré, John M.....325 West End Ave., New York, N. Y.
- \*1926 Lorie, Alvin J.....Commerce Bldg., Kansas City, Mo.
- 1910 Loughran, Robert L.....Sharon, Conn.
- \*1924 Lukens, Robert M.....1923 Spruce St., Philadelphia, Pa.
- 1934 Lupton, Irving M.....Medical Arts Bldg., Portland, Ore.
- \*1933 Lux, Paul.....Commerce Bldg., Kansas City, Mo.
- \*1919 Lyman, Harry W.....Carleton Bldg., St. Louis, Mo.
- \*1923 Lyons, Horace R.....30 N. Michigan Ave., Chicago, Ill.
- \*1915 Mackenzie, George W.,...269 S. 19th St., Philadelphia, Pa.
- 1920 MacLachlan, A. A.....May Bldg., Pittsburgh, Pa.
- 1918 Maclay, Otis H.....30 N. Michigan Ave., Chicago, Ill.
- 1907 Macpherson, Duncan.....140 E. 54th St., New York, N. Y.
- 1896 Malcolm, Percy E. D.....410 Park Ave., New York, N. Y.
- \*1923 Marshall, Watson.....121 University Pl., Pittsburgh, Pa.
- 1931 Martin, Robert C.....384 Post St., San Francisco, Cal.
- 1905 Mason, William Beverly....1738 M. St., Washington, D. C.
- 1928 Matheson, J. P.....6 W. Seventh St., Charlotte, N. C.
- 1912 Matthews, Justus.....Metro. Bk. Bldg., Minneapolis, Minn.
- 1921 Maybaum, Jacob L.....1070 Park Ave., New York, N. Y.
- 1926 McAskill, James E....Woolworth Bldg., Watertown, N. Y.
- \*1925 McCarthy, Merrick F...Union Central Bldg., Cincinnati, O.
- \*1933 McClellan, Wilburt E..50 Farmington Ave., Hartford, Conn.
- 1932 McCollough, Thomas B...121 University Pl., Pittsburgh, Pa.
- 1915 McCready, James H.....121 University Pl., Pittsburgh, Pa.
- \*1907 McCullagh, Samuel.....2 E. 54th St., New York, N. Y.
- 1929 McDougall, Calhoun.....Medical Arts Bldg., Atlanta, Ga.
- 1935 McDougall, William L.....Doctors' Bldg., Atlanta, Ga.
- 1897 McKernon, James F.....835 Fifth Ave., New York, N. Y.
- 1902 McKimmie, O. A. M...1301 Mass. Ave., Washington, D. C.
- 1915 McKinney, Richmond..1052 Madison Ave., Memphis, Tenn.
- 1935 McLaurin, John G.....Medical Arts Bldg., Dallas, Texas
- \*1929 McMahon, Bernard J...634 N. Grand Blvd., St. Louis, Mo.

- \*1929 McMurray, John B. . . . . Wash. Tr. Bldg., Washington, Pa.
- 1935 McNichols, William A. . . . . National Bank Bldg., Dixon, Ill.
- \*1897 McReynolds, John O. . . . . Mercantile Bank Bldg., Dallas, Texas
- 1935 Meltzer, Philip E. . . . . 20 Charlesgate, W., Boston, Mass.
- 1926 Mercer, Richard E. . . . . David Whitney Bldg., Detroit, Mich.
- 1925 Merriman, M. Heminway. 115 Prospect St., Waterbury, Conn.
- 1896 Mial, Leonidas L. . . . . 38 Elm St., Morristown, N. J.
- \*1911 Miller, Clifton M. . . . . Stuart Circle Hsp., Richmond, Va.
- \*1934 Miller, John. . . . . 63 North St., Greenwich, Conn.
- 1914 Miller, H. Edward. . . . . 728 Missouri Bldg., St. Louis, Mo.
- \*1925 Mithoefer, William. . . . . 19 Garfield Pl., Cincinnati, Ohio
- 1935 Montgomery, Robert R. . . . . Security Bldg., Long Beach, Cal.
- 1905 Mooney, James J. . . . . 226 Linwood Ave., Buffalo, N. Y.
- 1929 Moore, Roy S. . . . . Medical Arts Bldg., Syracuse, N. Y.
- \*1903 Moore, T. W. . . . . 1050 Fifth Ave., Huntington, W. Va.
- 1924 Moore, William F. . . . . 255 S. 16th St., Philadelphia, Pa.
- \*1915 Moorhead, Robert L. . . . . 125 Remsen St., Brooklyn, N. Y.
- 1931 Moriarty, Richard W. . . . . Medical Center, White Plains, N. Y.
- \*1922 Morris, Austin G. . . . . 277 Alexander St., Rochester, N. Y.
- 1931 Morrison, W. Wallace. . . . . 39 E. 50th St., New York, N. Y.
- 1925 Morse, Ashley W. . . . . Butte, Mont.
- \*1903 Mosher, Harris P. . . . . 828 Beacon St., Boston, Mass.
- 1927 Muckleston, H. S. . . . . Hollywood Blvd., Los Angeles, Cal.
- 1895 Munger, Carl E. . . . . 85 Grove St., Waterbury, Conn.
- 1932 Murphy, Albert B. . . . . 2830 Grand Ave., Everett, Wash.
- \*1924 Murphy, Grover E. . . . . Woodward Bldg., Birmingham, Ala.
- 1900 Murray, Gilbert D. 200 Worthington Ave., Spring Lake, N. J.
- \*1901 Myers, Harry L. . . . . Medical Arts Bldg., Norfolk, Va.
- 1926 Myerson, Mervin C. . . . . 134 E. 64th St., New York, N. Y.
- 1895 Myles, Robert C. . . . . 1075 Park Ave., New York, N. Y.
- \*1924 Naftzger, Jesse B. . . . . Davidson Bldg., Sioux City, Iowa
- \*1930 Nash, C. Stewart. . . . . 277 Alexander St., Rochester, N. Y.
- \*1918 New, Gordon B. . . . . Mayo Foundation, Rochester, Minn.
- 1924 Newhart, Horace. . . . . Medical Arts Bldg., Minneapolis, Minn.
- \*1927 Novak, Frank J., Jr. . . . . 30 N. Michigan Ave., Chicago, Ill.
- \*1924 Orton, Henry B. . . . . 24 Commerce St., Newark, N. J.
- 1920 Ostrom, Louis. . . . . Mfr. Trust Bldg., Rock Island, Ill.
- 1908 Page, John R. . . . . 127 E. 62nd St., New York, N. Y.
- 1935 Palmer, Arthur. . . . . 667 Madison Ave., New York, N. Y.
- 1934 Pearlman, Samuel J. . . . . 180 N. Michigan Ave., Chicago, Ill.
- 1920 Pearson, William W. . . . . Bankers Tr. Bldg., Des Moines, Iowa
- \*1930 Phelps, Kenneth A. . . . . Med. Arts Bldg., Minneapolis, Minn.

- \*1926 Phillips, Frank L.....405 Temple St., New Haven, Conn.
- 1896 Pierce, Norval H.....Del Mar, Cal.
- 1932 Pinkerton, Forrest J.....Young Bldg., Honolulu, T. H.
- \*1923 Pitkin, Carlos E....Carnegie Medical Bldg., Cleveland, Ohio
- \*1928 Poirier, George H.....60 Bay State Rd., Boston, Mass.
- \*1923 Pollock, H. L.....2551 N. Clark St., Chicago, Ill.
- 1933 Pond, Casper W.....Kane Bldg., Pocatello, Idaho
- \*1922 Porter, Charles T....520 Commonwealth Ave., Boston, Mass.
- 1926 Porter, Lewis B.....195 Thayer St., Providence, R. I.
- 1924 Potts, John B.....Medical Arts Bldg., Omaha, Neb.
- 1931 Preston, H. Grant...Professional Bldg., Harrisonburg, Va.
- 1924 Proetz, Arthur W.....Beaumont Bldg., St. Louis, Mo.
- 1899 Rae, John B.....34 E. 60th St., New York, N. Y.
- 1920 Reeder, James E.....Davidson Bldg., Sioux City, Iowa
- \*1931 Richards, Lyman G....319 Longwood Ave., Boston, Mass.
- 1923 Ridpath, Robert F..18th and Chestnut Sts., Philadelphia, Pa.
- \*1929 Roberts, Edward R.....Medical Bldg., Bridgeport, Conn.
- 1928 Roberts, Sam E.....Professional Bldg., Kansas City, Mo.
- 1905 Roberts, Walter.....1921 Spruce St., Philadelphia, Pa.
- 1907 Robinson, John A.....30 W. 59th St., New York, N. Y.
- 1896 Root, Arthur G.....218 State St., Albany, N. Y.
- 1934 Rosenberger, Harry C..Carnegie Med. Bldg., Cleveland, Ohio
- 1908 Rosenheim, Sylvan.....Medical Arts Bldg., Baltimore, Md.
- 1896 Ross, George T.....1469 Drummond St., Montreal, Canada
- \*1895 Roy, Dunbar.....Grand Opera House, Atlanta, Ga.
- \*1925 Salinger, Samuel.....25 E. Washington St., Chicago, Ill.
- 1904 Sandels, C. C.....Westinghouse Bldg., Pittsburgh, Pa.
- 1907 Sauer, William E....3720 Washington Blvd., St. Louis, Mo.
- 1922 Sautter, Carl M.....40 E. 61st St., New York, N. Y.
- \*1928 Schall, LeRoy A....270 Commonwealth Ave., Boston, Mass.
- 1921 Schipfer, Lloyd A.....952 N. Michigan Ave., Chicago, Ill.
- 1923 Scholz, Roy P.....Metropolitan Bldg., St. Louis, Mo.
- \*1933 Seaver, Edwin P., Jr...227 Union St., New Bedford, Mass.
- 1921 Selfridge, Grant.....Fitzhugh Bldg., San Francisco, Cal.
- 1914 Sensensy, Eugene T.....Lister Bldg., St. Louis, Mo.
- \*1922 Sewall, Edward C..Stanford Univ. Hsp., San Francisco, Cal.
- \*1922 Seydell, Ernest M....1st National Bank Bldg., Wichita, Kan.
- \*1921 Sharrett, George O.....119 Bedford St., Cumberland, Md.
- 1898 Shattuck, Warren S.....160 Clinton St., Brooklyn, N. Y.
- \*1922 Shea, John J.....1018 Madison Ave., Memphis, Tenn.
- \*1907 Shurly, Burt R.....62 W. Adams Ave., Detroit, Mich.
- \*1932 Shuster, Benjamin H...1930 Chestnut St., Philadelphia, Pa.



- 1935 Schuster, Franklin P...1st Nat'l Bank Bldg., El Paso, Texas  
 1935 Schuster, Stephan A...1st Nat'l Bank Bldg., El Paso, Texas  
 1915 Simpson, John R.....Medical Arts Bldg., Pittsburgh, Pa.  
 \*1922 Simpson, W. Likely..Phys. and Surgs. Bldg., Memphis, Tenn.  
 \*1927 Skillern, Samuel R., Jr.....1734 Pine St., Philadelphia, Pa.  
 1908 Smith, Angelo J.....207 Park Ave., Yonkers, N. Y.  
 \*1925 Smith, Carroll.....Paulsen Bldg., Spokane, Wash.  
 1928 Smith, Clarence H.....140 E. 54th St., New York, N. Y.  
 1905 Smith, E. Terry.....36 Pearl St., Hartford, Conn.  
 1935 Smith, J. Morrisset.....123 E. 53rd St., New York, N. Y.  
 \*1921 Sonnenschein, Robert...180 N. Michigan Ave., Chicago, Ill.  
 \*1913 Spencer, Frank R.....Physicians' Bldg., Boulder, Colo.  
 1911 Sperry, Frederick N...85 Whitney Ave., New Haven, Conn.  
 1906 Spratt, Charles N....900 Nicollet Ave., Minneapolis, Minn.  
 \*1928 Sprowl, Frederic G....Medical Arts Bldg., Spokane, Wash.  
 \*1932 Stark, W. Berkeley....Medical Arts Bldg., Toronto, Canada  
 1915 Stein, Edgar J.....439 N. Duke St., Lancaster, Pa.  
 1900 Stein, Otto J.....Palos Verdes Ests., Cal.  
 1932 Stevenson, H. M....421 Huguenot St., New Rochelle, N. Y.  
 1930 Stevenson, Walter D....904 W. C. U. Bldg., Quincy, Ill.  
 1915 Stickle, Charles W.....86 Pierrepont St., Brooklyn, N. Y.  
 1931 Stiles, Porter.....Medical Arts Bldg., Birmingham, Ala.  
 1902 Stillman, F. L.....185 E. State St., Columbus, Ohio  
 1935 Stout, Philip S.....269 S. 19th St., Philadelphia, Pa.  
 \*1922 Strauss, Jerome F.....104 S. Michigan Ave., Chicago, Ill.  
 1915 Sullivan, John J., Jr.....Scranton Life Bldg., Scranton, Pa.  
 \*1933 Sulzman, Frank M.....1831 Fifth St., Troy, N. Y.  
 \*1923 Taylor, H. Marshall...111 W. Adams St., Jacksonville, Fla.  
 1935 Tebbutt, Harry K.....240 State St., Albany, N. Y.  
 \*1931 Theobald, Walter H...307 N. Michigan Ave., Chicago, Ill.  
 1895 Thigpen, Charles A....401 S. Court St., Montgomery, Ala.  
 1923 Tholen, Emil F.....1136 W. Sixth St., Los Angeles, Cal.  
 1927 Thompson, George H.....74 North St., Pittsfield, Mass.  
 \*1923 Thompson, Howard E.....1100 Main St., Dubuque, Iowa  
 1907 Thomson, John J.....531 Lincoln Ave., Mt. Vernon, N. Y.  
 \*1921 Tobey, George L., Jr..270 Commonw'th Ave., Boston, Mass.  
 \*1922 Tobey, Harold G...270 Commonwealth Ave., Boston, Mass.  
 1905 Trowbridge, D. H.....Fresno, Cal.  
 \*1924 Tucker, Gabriel.....326 S. 19th St., Philadelphia, Pa.  
 1923 Unger, Max.....226 W. 42nd St., New York, N. Y.  
 \*1926 Uren, Claude T....City National Bank Bldg., Omaha, Neb.  
 1915 Vanderhoof, Don A..Exc. Nat. Bk. Bldg., Colo. Sprgs., Colo.

- 1934 Van Poole, Gideon McD. . . . . Young Bldg., Honolulu, T. H.  
 1932 Viole, Pierre. . . . . 1930 Wilshire Blvd., Los Angeles, Cal.  
 \*1905 Voislowsky, Antonie P. . . . . 33 E. 68th St., New York, N. Y.  
 1915 Voorhees, Irving W. . . . . 140 E. 54th St., New York, N. Y.  
 \*1907 Wales, Ernest DeWolf. . Med. Arts Bldg., Indianapolis, Ind.  
 \*1921 Walker, D. Harold. . . . . 5 Bay State Rd., Boston, Mass.  
 1935 Wallerstein, Emanuel U. . Professional Bldg., Richmond, Va.  
 1931 Waltz, Manford R. . . . . Med. and Den. Bldg., Seattle, Wash.  
 1924 Wanamaker, Allison T. . . 817 Summit Ave., Seattle, Wash.  
 1932 Warren, William C., Jr. . . . 478 Peach Tree St., Atlanta, Ga.  
 1931 Warwick, Harold L. . . . . Med. Arts Bldg., Fort Worth, Texas  
 1930 Weih, Elmer P. . . . . 605 Wilson Bldg., Clinton, Iowa  
 1914 Weil, Arthur I. . . . . Phys. and Surgs. Bldg., New Orleans, La.  
 \*1921 Weinberger, N. S. . . . . Robert Packer Hsp., Sayre, Pa.  
 1901 Wells, Walter A. . . . . 1606 20th St., N.W., Washington, D. C.  
 1925 Westlake, Samuel B. . . 3720 Washington Ave., St. Louis, Mo.  
 1934 Whelan, George L. . . . . 2100 Walnut St., Philadelphia, Pa.  
 \*1924 Wherry, W. P. . . . . 1500 Med. Arts Bldg., Omaha, Neb.  
 \*1920 White, Francis W. . . . . 174 W. 58th St., New York, N. Y.  
 \*1925 White, J. Warren. . . . . Medical Arts Bldg., Norfolk, Va.  
 \*1923 Willcutt, George H. . . . . Albert Bldg., San Rafael, Calif.  
 \*1932 Williams, Horace J. . . . . 5908 Green St., Germantown, Pa.  
 1921 Wilson, J. Gordon. . . . . 104 S. Michigan Ave., Chicago, Ill.  
 \*1925 Winter, George E. . . . . Roger Bldg., Jackson, Mich.  
 1935 Wood, Earl LeRoy. . . . . 192 Roseville Ave., Newark, N. J.  
 1906 Wood, George B. . . . . Physicians' Bldg., Philadelphia, Pa.  
 \*1927 Wood, Vivian V. . . . . 3720 Washington Ave., St. Louis, Mo.  
 \*1929 Woodward, Fletcher D. . . . P. O. Box 1685, University, Va.  
 \*1898 Woodward, John F. . . . . 528 Redgate Ave., Norfolk, Va.  
 \*1932 Zinkhan, Arthur M. . . . The Rochambeau, Washington, D. C.  
 \*1927 Zinn, Waitman F. . . . . Medical Arts Bldg., Baltimore, Md.

## EMERITUS FELLOWS

- 1905 Bane, William C. . . . . 1612 Tremont St., Denver, Colo.  
 \*1904 Barnhill, John F. . . . . 5369 Pine Tree Dr., Miami Beach, Fla.  
 1906 Borden, Charles R. C. . . . 1759 Beacon St., Brookline, Mass.  
 1898 Brown, J. Price. . . . . 15 Glebe Rd., W., Toronto, Canada  
 1897 Cobb, Frederick C. . . . . Bradentown, Fla.  
 1895 Dunn, John. . . . . 411 E. Franklin St., Richmond, Va.  
 1907 Emerson, Francis P. . . . . Park Rd., Franklin, Mass.

1907	Emerson, Linn.....	Metropolitan Bldg., Orange, N. J.
1900	Hartz, Henry J.....	Whitney Bldg., Detroit, Mich.
1900	Hopkins, F. E.....	146 Chestnut St., Springfield, Mass.
1901	Hubbard, Thomas.....	525 Winthrop St., Toledo, Ohio
1900	Ingersoll, John M.....	1700 S. Bay Shore Lane, Miami, Fla.
1895	Jack, Frederick L.....	215 Beacon St., Boston, Mass.
1905	Kerrison, Philip D.....	10 E. 43rd St., New York, N. Y.
1912	Lockard, Lorenzo B.....	655 Gaylord St., Denver, Colo.
1900	McCaw, James F.....	Woolworth Bldg., Watertown, N. Y.
1904	Martinez, Emilio..	Calle H., No. 144—Vedado, Havana, Cuba
1901	Moss, Robert E.....	La Grange, Texas
1900	Packard, Francis R.....	302 S. 19th St., Philadelphia, Pa.
1906	Pischel, Kaspar.....	490 Post St., San Francisco, Cal.
1906	Reik, H. O.....	Vermont Apts., Atlantic City, N. J.
1901	Roberts, W. Humes..	505 Professional Bldg., Pasadena, Cal.
1917	Shambaugh, George E..	122 S. Michigan Ave., Chicago, Ill.
1895	Thompson, John A.....	R. F. D. No. 4, Brookville, Ind.
1896	Wagner, Henry L.....	518 Sutter St., San Francisco, Cal.
1895	White, Joseph A.....	200 E. Franklin St., Richmond, Va.

## HONORARY FELLOWS

1907	Ballance, C. E., Sir.....	106 Harley St., W. I., London, Eng.
1929	Delavan, D. Bryson.....	30 E. 60th St., New York, N. Y.
1900	Dundas-Grant, Sir James	148 Harley St., W. I., London, Eng.
1923	Fraser, John S.....	50 Wimpole St., Edinburgh, Scot.
1919	Lemaitre, Fernand....	120 Ave., Victor Hugo, Paris, France
1909	Love, James Kerr.....	5 Newton Pl., Glasgow, C. 3, Scot.
1898	Schmieglow, E.....	18 Norregade, Copenhagen, Den.
1919	Thomson, Sir St. Clair.	64 Wimpole St., W. I., London, Eng.
1902	Turner, Logan.....	27 Walker St., Edinburgh, Scot.

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J. A. Thompson, M.D.
Max Thorne, M.D.
H. S. Straight, M.D.
N. H. Pierce, M.D.
H. W. Loeb, M.D.
L. C. Cline, M.D.
C. Jackson, M.D.
Thomas Hubbard, M.D.
W. L. Ballenger, M.D.
J. M. Ingersoll, M.D.
J. F. Barnhill, M.D.
R. B. Canfield, M.D.
H. J. Hartz, M.D.
Latayette Page, M.D.
John A. Thompson, M.D.
Wm. E. Sauer, M.D.
B. R. Shurly, M.D.
William B. Chamberlin, M.D.
John E. Brown, M.D.
D. W. Layman, M.D.
Otto J. Stein, M.D.
Samuel Iglauer, M.D.
Joseph C. Beck, M.D.
Greenfield Sluder, M.D.
Gordon B. New, M.D.
Ray Connor, M.D.
G. F. Keiper, M.D.
Lloyd A. Schipfer, M.D.
Harry W. Lyman, M.D.
Gordon F. Harkness, M.D.
Robert Sonnenschein, M.D.
Harold I. Lillie, M.D.
Albert C. Furstenberg, M.D.
Austin A. Hayden, M.D.
Edward King, M.D.
Frank J. Novak, Jr., M.D.
John W. Carmack, M.D.
William E. Grove, M.D.

1931	Claude T. Uren, M.D.
1932	Alvin G. Lorie, M.D.
1933	Dean M. Lierle, M.D.
1934	Arthur W. Proetz, M.D.
1935	Edward H. Cary, M.D.
1936	Harry W. Lyman, M.D.

## Western Section

J. E. Logan, M.D.
J. E. Schadle, M.D.
H. L. Warner, M.D.
Robert Levy, M.D.
J. O. McReynolds, M.D.
E. W. Fleming, M.D.
P. F. Gildea, M.D.
R. W. Payne, M.D.
M. A. Goldstein, M.D.
H. B. Ellis, M.D.
W. C. Bare, M.D.
W. H. Roberts, M.D.
J. C. Beck, M.D.
A. R. Solenberger, M.D.
Wm. H. Dudley, M.D.
Thomas J. Gallagher, M.D.
Hill Hastings, M.D.
Lorenzo B. Lockard, M.D.
Frank R. Spencer, M.D.
Thomas E. Carmody, M.D.
James A. Patterson, M.D.
H. B. Graham, M.D.
John J. Kyle, M.D.
H. Bert Ellis, M.D.
Hill Hastings, M.D.
Grant Selfridge, M.D.
Isaac H. Jones, M.D.
Eugene K. Lewis, M.D.
E. C. Sewall, M.D.
J. Mackenzie Brown, M.D.
Frank B. Kistner, M.D.
Ralph A. Fenton, M.D.
Henry M. Cunningham, M.D.
George H. Willcutt, M.D.
Allison T. Wanmaker, M.D.
Harold A. Fletcher, M.D.
Robert R. Hampton, M.D.
Ralph F. Davis, M.D.
Frank A. Burton, M.D.
Carroll Smith, M.D.



## SECRETARY

- 1896 to 1900  
Robert C. Myles, M.D.  
1901 to 1906  
Wendell C. Phillips, M.D.  
1907 to 1916  
Thomas J. Harris, M.D.  
1917 to 1924  
William H. Haskin, M.D.  
1925 to date  
Robert L. Loughran, M.D.

## TREASURER

- 1896 to 1900  
Robert C. Myles, M.D.  
1901 to 1907  
Ewing W. Day, M.D.  
1908—Chevalier Jackson, M.D.  
1909 to date  
Ewing W. Day, M.D.  
EDITORS  
1919 to 1933  
George L. Richards, M.D.  
1933 to date  
Lyman G. Richards, M.D.

## COUNCIL

- 1896—T. P. Berens, M.D.  
J. H. Billings, M.D.  
D. L. Hubbard, M.D.  
H. H. Curtis, M.D.  
W. C. Phillips, M.D.  
R. C. Myles, M.D.  
1897—G. H. Makuen, M.D.  
T. P. Berens, M.D.  
W. C. Phillips, M.D.  
H. H. Curtis, M.D.  
E. W. Day, M.D.  
D. L. Hubbard, M.D.  
1898—J. E. Nichols, M.D.  
H. H. Curtis, M.D.  
L. A. Coffin, M.D.  
E. W. Day, M.D.  
W. C. Phillips, M.D.  
C. W. Richardson, M.D.  
S. E. Solly, M.D.  
1899—W. H. Daly, M.D.  
W. C. Phillips, M.D.  
T. H. Halsted, M.D.  
J. O. Roe, M.D.  
H. W. Loeb, M.D.  
T. C. Christy, M.D.  
F. J. Jack, M.D.  
D. L. Hubbard, M.D.  
1900—S. E. Solly, M.D.  
W. H. Daley, M.D.  
A. W. Calhoun, M.D.  
J. B. Clemens, M.D.  
J. F. McKernon, M.D.  
W. C. Phillips, M.D.  
J. E. Sheppard, M.D.  
J. Price Brown, M.D.  
Max Thorne, M.D.  
1901—D. B. Kyle, M.D.  
S. MacCuen Smith, M.D.  
S. E. Solly, M.D.  
J. Price Brown, M.D.  
W. H. Daley, M.D.  
J. B. Clemens, M.D.  
J. F. McKernon, M.D.  
C. W. Richardson, M.D.  
F. C. Cobb, M.D.  
1902—D. B. Kyle, M.D.  
S. E. Solly, M.D.  
J. F. McKernon, M.D.  
S. MacCuen Smith, M.D.  
F. C. Cobb, M.D.  
N. H. Pierce, M.D.  
T. P. Berens, M.D.  
J. A. White, M.D.  
1903—C. W. Richardson, M.D.  
R. C. Myles, M.D.  
D. B. Kyle, M.D.  
J. F. McKernon, M.D.  
S. MacCuen Smith, M.D.  
F. C. Cobb, M.D.  
N. H. Pierce, M.D.  
T. P. Berens, M.D.  
C. R. Holmes, M.D.  
1904—J. A. Stucky, M.D.  
C. W. Richardson, M.D.  
R. C. Myles, M.D.  
A. G. Root, M.D.  
T. P. Berens, M.D.  
C. R. Holmes, M.D.  
E. B. Dench, M.D.  
F. L. Jack, M.D.  
J. E. Schadle, M.D.  
F. R. Packard, M.D.  
1905—N. H. Pierce, M.D.  
J. A. Stucky, M.D.  
C. W. Richardson, M.D.  
L. A. Coffin, M.D.  
T. H. Halsted, M.D.  
C. R. Holmes, M.D.  
E. B. Dench, M.D.  
F. L. Jack, M.D.  
J. E. Schadle, M.D.  
F. R. Packard, M.D.

- 1906—F. C. Cobb, M.D.  
N. H. Pierce, M.D.  
J. A. Stucky, M.D.  
E. B. Dench, M. D.  
F. L. Jack, M.D.  
F. R. Packard, M.D.  
L. A. Coffin, M.D.  
T. H. Halsted, M.D.  
J. F. McKernon, M.D.  
H. W. Loeb, M.D.
- 1907—J. E. Logan, M.D.  
F. C. Cobb, M.D.  
N. H. Pierce, M.D.  
L. A. Coffin, M.D.  
T. H. Halsted, M.D.  
J. F. McKernon, M.D.  
H. W. Loeb, M.D.  
C. W. Richardson, M.D.  
D. B. Kyle, M.D.
- 1908—W. C. Phillips, M.D.  
J. E. Logan, M.D.  
F. C. Cobb, M.D.  
J. F. McKernon, M.D.  
H. W. Loeb, M.D.  
C. W. Richardson, M.D.  
D. B. Kyle, M.D.  
C. R. Holmes, M.D.  
J. F. McCaw, M.D.
- 1909—J. E. Logan, M.D.  
D. B. Kyle, M.D.  
C. W. Richardson, M.D.  
W. C. Phillips, M.D.  
J. F. McCaw, M.D.  
W. R. Lincoln, M.D.  
G. L. Richards, M.D.  
J. F. Barnhill, M.D.  
G. H. Makuen, M.D.
- 1910—C. R. Holmes, M.D.  
G. L. Richards, M.D.  
J. F. Barnhill, M.D.  
G. H. Makuen, M.D.  
N. H. Pierce, M.D.  
W. C. Phillips, M.D.  
F. R. Packard, M.D.  
A. B. Duel, M.D.  
Robert Levy, M.D.
- 1911—J. F. McKernon, M.D.  
J. A. White, M.D.  
N. L. Wilson, M.D.  
C. R. Holmes, M.D.  
A. B. Duel, M.D.  
F. R. Packard, M.D.  
Robert Levy, M.D.  
G. L. Richards, M.D.  
N. H. Pierce, M.D.
- 1912—C. R. Holmes, M.D.  
F. R. Packard, M.D.  
N. H. Pierce, M.D.  
J. F. McKernon, M.D.  
J. A. White, M.D.  
N. L. Wilson, M.D.  
C. Jackson, M.D.  
M. A. Goldstein, M.D.  
A. B. Duel, M.D.
- 1913—James F. McKernon, M.D.  
Joseph A. White, M.D.  
Norton L. Wilson, M.D.  
Chevalier Jackson, M.D.  
Max A. Goldstein, M.D.  
Arthur B. Duel, M.D.  
Harris P. Mosher, M.D.  
E. Fletcher Ingals, M.D.  
G. H. Makuen, M.D.
- 1914—Chevalier Jackson, M.D.  
Max A. Goldstein, M.D.  
Arthur B. Duel, M.D.  
Harris P. Mosher, M.D.  
E. Fletcher Ingals, M.D.  
G. Hudson-Makuen, M.D.  
H. Holbrook Curtis, M.D.  
John E. Sheppard, M.D.  
Robert Levy, M.D.
- 1915—Joseph A. White, M.D.  
Harris P. Mosher, M.D.  
E. Fletcher Ingals, M.D.  
G. Hudson-Makuen, M.D.  
H. Holbrook Curtis, M.D.  
John E. Sheppard, M.D.  
Robert Milligan, M.D.  
John M. Ingersoll, M.D.  
B. R. Shurly, M.D.
- 1916—G. Hudson-Makuen, M. D.  
H. Holbrook Curtis, M.D.  
John E. Sheppard, M.D.  
Robert Milligan, M.D.  
John M. Ingersoll, M.D.  
B. R. Shurly, M.D.  
E. B. Dench, M.D.  
D. J. Gibb Wishart, M.D.  
Robert Levy, M.D.
- 1917—Robert Milligan, M.D.  
John M. Ingersoll, M.D.  
E. B. Dench, M.D.  
D. J. Gibb Wishart, M.D.  
B. R. Shurly, M.D.  
Robert Levy, M.D.  
S. MacCuen Smith, M.D.  
Thomas H. Halsted, M.D.  
Walter A. Wells, M.D.

- 1918—D. J. Gibbs<sup>(1)</sup> Wishart, M.D.  
B. R. Shurly, M.D.  
Robert Levy, M.D.  
S. MacCuen Smith, M.D.  
Thomas H. Halsted, M.D.  
Walter A. Wells, M.D.  
T. J. Harris, M.D.  
Francis P. Emerson, M.D.  
L. W. Dean, M.D.
- 1919—S. MacCuen Smith, M.D.  
Thomas H. Halsted, M.D.  
Walter A. Wells, M.D.  
Thomas J. Harris, M.D.  
Francis P. Emerson, M.D.  
L. W. Dean, M.D.  
Frank R. Spencer, M.D.  
Joseph C. Beck, M.D.  
Wendell C. Phillips, M.D.
- 1920—Thomas J. Harris, M.D.  
Lee Wallace Dean, M.D.  
Francis P. Emerson, M.D.  
George L. Richards, M.D.  
Joseph C. Beck, M.D.  
Frank R. Spencer, M.D.  
Wendell C. Phillips, M.D.  
Herbert S. Birkett, M.D.  
Lafayette Page, M.D.  
Robert C. Lynch, M.D.
- 1921—George L. Richards, M.D.  
George E. Shambaugh, M.D.  
Frank R. Spencer, M.D.  
Wendell C. Phillips, M.D.  
Herbert S. Birkett, M.D.  
Lafayette Page, M.D.  
Robert C. Lynch, M.D.  
Harris P. Mosher, M.D.  
Joseph F. McCaw, M.D.  
Dunbar Roy, M.D.
- 1922—Herbert S. Birkett, M.D.  
Lafayette Page, M.D.  
Robert C. Lynch, M.D.  
Harris P. Mosher, M.D.  
Joseph F. McCaw, M.D.  
Dunbar Roy, M.D.  
Lee Wallace Dean, M.D.  
James F. McKernon, M.D.  
Norval H. Pierce, M.D.
- 1923—Harris P. Mosher, M.D.  
J. F. McCaw, M.D.  
J. W. Jerve, M.D.  
Lee Wallace Dean, M.D.  
J. F. McKernon, M.D.  
N. H. Pierce, M.D.  
L. A. Coffin, M.D.  
B. R. Shurly, M.D.  
R. H. Skillern, M.D.
- 1924—Lee W. Dean, M.D.  
James F. McKernon, M.D.  
Norval H. Pierce, M.D.  
Lewis A. Coffin, M.D.  
B. R. Shurly, M.D.  
R. H. Skillern, M.D.  
Dunbar Roy, M.D.  
William V. Mullin, M.D.  
Leon E. White, M.D.
- 1925—Lewis A. Coffin, M.D.  
B. R. Shurly, M.D.  
R. H. Skillern, M.D.  
Dunbar Roy, M.D.  
William V. Mullin, M.D.  
Leon E. White, M.D.  
H. W. Loeb, M.D.  
R. A. Fenton, M.D.  
R. C. Lynch, M.D.
- 1926—Dunbar Roy, M.D.  
William V. Mullin, M.D.  
Leon E. White, M.D.  
Class B.  
Hanau W. Loeb, M.D.  
Ralph A. Fenton, M.D.  
Robert C. Lynch, M.D.  
William H. Haskin, M.D.  
Harold I. Lillie, M.D.  
Henry Hall Forbes, M.D.
- 1927—Hanau W. Loeb, M.D.  
Ralph A. Fenton, M.D.  
Robert C. Lynch, M.D.  
William H. Haskin, M.D.  
Harold I. Lillie, M.D.  
Henry Hall Forbes, M.D.  
John M. Ingersoll, M.D.  
Joseph B. Greene, M.D.  
Gordon Berry, M.D.
- 1928—William H. Haskin, M.D.  
Harold I. Lillie, M.D.  
Henry Hall Forbes, M.D.  
John M. Ingersoll, M.D.  
Joseph B. Greene, M.D.  
Gordon Berry, M.D.  
Burt R. Shurly, M.D.  
Edward C. Sewall, M.D.  
Frank L. Dennis, M.D.
- 1929—John M. Ingersoll, M.D.  
Joseph B. Greene, M.D.  
Gordon Berry, M.D.  
Burt R. Shurly, M.D.  
Edward C. Sewall, M.D.  
Frank L. Dennis, M.D.  
John F. Barnhill, M.D.  
Thomas E. Carmody, M.D.  
Antonie P. Voislowsky, M.D.

- 1930—Burt R. Shurly, M.D.  
 Edward C. Sewall, M.D.  
 Frank L. Dennis, M.D.  
 John F. Barnhill, M.D.  
 Thomas E. Carmody, M.D.  
 Antonie P. Voislavsky, M.D.  
 Hill Hastings, M.D.  
 D. C. Jarvis, M.D.  
 J. W. Jervy, M.D.
- 1931—John F. Barnhill, M.D.  
 Thomas E. Carmody, M.D.  
 Antonie P. Voislavsky, M.D.  
 Hill Hastings, M.D.  
 D. C. Jarvis, M.D.  
 J. W. Jervy, M.D.  
 Ross H. Skillern, M.D.  
 W. P. Wherry, M.D.  
 George E. Shambaugh, M.D.  
 Wm. H. Haskin, M.D.
- 1932—Hill Hastings, M.D.  
 D. C. Jarvis, M.D.  
 J. W. Jervy, M.D.  
 William H. Haskin, M.D.  
 Harold I. Lillie, M.D.  
 W. P. Wherry, M.D.  
 Joseph C. Beck, M.D.  
 Perry G. Goldsmith, M.D.  
 Max A. Goldstein, M.D.
- 1933—William H. Haskin, M.D.  
 Harold I. Lillie, M.D.  
 W. P. Wherry, M.D.  
 William B. Chamberlin, M.D.
- Perry G. Goldsmith, M.D.  
 Max A. Goldstein, M.D.  
 Edmund Prince Fowler, M.D.  
 Clifton M. Miller, M.D.  
 Harris P. Mosher, M.D.
- 1934—William B. Chamberlin, M.D.  
 Perry G. Goldsmith, M.D.  
 Max A. Goldstein, M.D.  
 Edmund Prince Fowler, M.D.  
 Clifton M. Miller, M.D.  
 Harris P. Mosher, M.D.  
 Joseph C. Beck, M.D.  
 Harrington B. Graham, M.D.  
 Harold I. Lillie, M.D.
- 1935—Edmund Prince Fowler, M.D.  
 Clifton M. Miller, M.D.  
 Harris P. Mosher, M.D.  
 Joseph C. Beck, M.D.  
 Harrington B. Graham, M.D.  
 Harold I. Lillie, M.D.  
 D. C. Jarvis, M.D.  
 J. W. Jervy, M.D.  
 W. P. Wherry, M.D.
- 1936—Joseph C. Beck, M.D.  
 Harold I. Lillie, M.D.  
 Harrington B. Graham, M.D.  
 J. W. Jervy, M.D.  
 D. C. Jarvis, M.D.  
 W. P. Wherry, M.D.  
 Lee M. Hurd, M.D.  
 Don M. Campbell, M.D.  
 Perry G. Goldsmith, M.D.



## DECEASED WHILE MEMBERS OF THE SOCIETY

## ACTIVE FELLOWS

Died	Elected	Died	Elected
1896 Noyes, J. I.....	1895	1921 Cott, George F.....	1904
1898 O'Dwyer, Joseph.....	1895	1921 Kuyk, D. A. ....	1906
1899 Hengst, D. A.....	1895	1921 Kyle, John F.....	1905
1899 Nichols, J. E.....	1895	1922 Lynah, Henry L.....	1915
1899 Thorner, Max.....	1895	1923 Edgar, Thomas O.....	1916
1900 Hopkins, Woolsey.....	1898	1923 Murphy, John W.....	1907
1900 Rankin, D. N.....	1897	1924 Berens, T. Passmore.....	1895
1900 Wenner, Ralph J.....	1898	1924 Bigelow, Frederick N.....	1918
1901 Daly, William H.....	1896	1924 Lowman, John B.....	1900
1901 Grady, L. B.....	1900	1925 Cox, Charles N.....	1896
1903 Chapman, S. Hartwell.....	1895	1925 Johnson, Walter B.....	1896
1903 Friedenber, Edward.....	1895	1925 Scarlett, Rufus B.....	1912
1903 Kibbe, A. B.....	1899	1926 Keiper, George F.....	1900
1905 Hoople, H. Nelson.....	1900	1926 McClelland, Lefferts A.....	1901
1906 Brandegee, William P.....	1898	1926 Hubbard, Ernest V.....	1917
1906 Solly, S. E.....	1895	1927 Robertson, Charles M.....	1919
1907 Harland, Wm. G. B.....	1905	1927 Finck, Harry P.....	1927
1908 Burnett, Peter V.....	1900	1927 Loeb, Hanau W.....	1895
1908 Peete, C. H.....	1904	1927 Chambers, Talbot R.....	1900
1908 Roosa, D. B. St. John.....	1895	1928 Logan, James E.....	1895
1908 Schadle, J. E.....	1895	1928 Kirkendall, J. S.....	1906
1909 Mullins, John B.....	1902	1928 Sluder, Greenfield.....	1911
1909 Sprague, Frank B.....	1896	1928 Vansant, Eugene L.....	1896
1910 Anderson, Willis S.....	1903	1928 White, Leon E.....	1918
1910 Calhoun, A. W.....	1895	1929 Banister, John M.....	1924
1910 Gradle, Henry.....	1906	1929 Ellegood, Joshua A.....	1913
1910 McGahan, Charles F.....	1900	1929 Guntzer, John H.....	1911
1911 Koyle, F. H.....	1897	1929 Page, Lafayette.....	1900
1912 Dudley, William F.....	1900	1929 Patterson, James A.....	1903
1912 Kimball, Irving E.....	1896	1929 Richardson, Charles W.....	1895
1913 Gildea, Patrick F.....	1897	1929 Sears, William H.....	1925
1914 Adams, John L.....	1895	1929 Smith, S. MacCuen.....	1895
1914 Cunningham, Frank M.....	1914	1929 Smyth, Herbert E.....	1902
1914 Gibb, Joseph S.....	1899	1930 Freudenthal, Wolff.....	1901
1914 Gruening, Emil.....	1905	1930 Waterman, James S.....	1900
1915 Mabon, John S.....	1895	1930 Cunningham, Henry M.....	1918
1915 Sheppard, John E.....	1895	1930 Skillern, Ross Hall.....	1910
1916 Ballenger, William L.....	1900	1930 Woodson, J. M.....	1926
1916 Kyle, D. Braden.....	1896	1931 Arrowsmith, Hubert.....	1903
1916 Roe, John O.....	1895	1931 Briggs, H. H.....	1914
1917 Dye, Herbert S.....	1902	1931 Campbell, William E.....	1903
1917 Makuen, G. Hudson.....	1896	1931 Demarest, Frederick F. C.....	1915
1917 Solenberger, Amos R.....	1901	1931 Lampe, Herman F.....	1926
1918 Chappell, Walter F.....	1908	1931 Levitt, Frederick C.....	1925
1918 Daly, Walter S.....	1906	1931 Lynch, Robert C.....	1913
1918 Holmes, Edgar M.....	1902	1931 Mackenty, John E.....	1911
1918 Ingals, E. Fletcher.....	1904	1931 Stucky, Joseph A.....	1895
1918 Yates, David G.....	1904	1931 Tucker, James C.....	1927
1919 Bird, Urban S.....	1915	1932 Campbell, William E.....	1903
1919 Blake, Clarence J.....	1907	1932 Canfield, R. Bishop.....	1904
1919 Kenefick, Joseph A.....	1899	1932 Crockett, Eugene A.....	1897
1919 Lutz, Stephen H.....	1900	1932 Fetteroff, George.....	1901
1919 Ray, James M.....	1919	1932 Fisher, Lewis.....	1918
1920 Curtis, H. Holbrook.....	1895	1932 Yankauer, Sidney.....	1909
1920 Friedberg, Stanton A.....	1914	1933 Connor, Ray.....	1914
1920 Holmes, Christian R.....	1900	1933 McCoy, John.....	1904
1920 Horn, Henry W.....	1915	1933 Oertel, Theodore E.....	1915
1920 Linhart, C. P.....	1899	1933 Steel, George E.....	1904

Died	Elected	Died	Elected
1934 Wishart, D. J. Gibb.....	1900	1934 Ziegelman, E. F.....	1932
1934 Dickson, John F.....	1929	1935 Carroll, George G.....	1934
1934 Thigpen, F. M.....	1895	1935 Dudley, William H.....	1900
1934 Mikell, P. V.....	1925	1935 Howard, Robert C.....	1930
1934 Carmack, John W.....	1929	1935 Renner, W. Scott.....	1895
1934 LaSalle, J. J.....	1908	1935 Mullin, William V.....	1919
1934 Phillips, W. C.....	1895	1935 Smyth, D. Campbell.....	1925
1934 Smith, Harmon.....	1903	1935 Webber, E. Russell.....	1923

## EMERITUS FELLOWS

1921 Ellis, H. Bert (Emer. 1921)	1899	1930 Worthington, Thomas C.	
1922 Leland, G. A. (Emer. 1921)	1904		(Emer. 1926) 1909
1927 Hyatt, Frank (Emer. 1921)	1895	1930 Vail, Derrick T.	
1927 Thrasher, A. B.			(Emer. 1924) 1906
	(Emer. 1921) 1896	1931 Wilson, Norton L.	
1927 Solis-Cohen, J.			(Emer. 1928) 1896
	(Emer. 1921) 1901	1935 Allport, Frank	
			(Emer. 1926) 1905

## HONORARY FELLOWS

1900 Whistler, W. MacNeill.....	1896	1921 Bell, Alex. Graham.....	1910
1901 Gougenheim, Achille.....	1896	1925 Luc, H. ....	1896
1902 Brown, Lenox.....	1896	1927 Magnus, R.....	1926
1902 Gruber, Joseph.....	1896	1928 MacIntyre, John.....	1896
1904 Smyly, Sir Philip.....	1896	1931 Mayer, Emil.....	1927
1912 Woakes, Edward.....	1896	1932 Law, Edward.....	1903
1915 Wolfenden, R. Norris.....	1896	1932 Gradenigo, Giuseppe.....	1907
1921 Semon, Sir Felix.....	1900	1932 Sendziak, J. ....	1898
1921 Joel, Joseph.....	1896	1934 Coakley, Cornelius G.....	1929











